Volume I

Section 8 - 13

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PRIVILEGED AND CONFIDENTIAL

PHILIP MORRIS
Briefing of Executives

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ATTORNEY WORK PRODUCT

PHILIP MORRIS

LEGISLATIVE BRIEFING MATERIALS

JULY, 1988

VOLUME I

TABS 8-13

Alexander Holtzman

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PHILIP MORRIS
LEGISLATIVE BRIEFING MATERIALS

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RESTRICTIONS ON ADVERTISING, PROMOTION AND MARKETING

At the hearing on H.R. 4543 before the Subcommittee on Transportation, Tourism and Hazardous Materials (the "Luken Committee") on June 8, 1988, some witnesses expressed concern about techniques of advertising and promotion purportedly used by some cigarette manufacturers -- distributing promotional cigarette samples to minors, sponsoring juvenile athletic contests and advertising on billboards directly facing centers of youth activity.

These criticisms are unfair. Philip Morris does not engage in such activities; and the specter of such abuses should not be used to restrict legitimate advertising by Philip Morris.

Sampling. Philip Morris neither encourages nor condones the promotional distribution of cigarette samples to minors. Along with the other manufacturers, Philip Morris subscribes to a Code of Sampling Practices that forbids sampling to persons under 21 years of age, and prohibits sampling in any public place within two blocks of a playground, school or other center of youth activities. Firms that sample for Philip Morris are required to observe these rules. For the employees of

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such firms, sampling to anyone under 21 is grounds for immediate discharge. Such misconduct can cost the sampling firm its contract with Philip Morris.

In addition to these voluntary prohibitions, many states already have laws banning the distribution of tobacco products to minors. Enforcement of these laws would readily solve any problems that actually may exist with respect to sampling to minors -- without banning sampling to adults. Cigarette sampling to adult smokers is an important promotional technique especially for introducing new brands and brand styles. The legislative history of the Public Health Cigarette Smoking Act of 1969 -- in which Congress enacted Section 1334(b) in its current form -- shows that sampling was recognized as an established form of cigarette "advertising or promotion." S. Rep. No. 566, 91st Cong., 1st Sess. 9 (1969); Cigarette Advertising and Labeling: Hearing on H.R. 6543 before the Consumer Subcomm. of the Senate Commerce Comm., 91st Cong., 1st Sess. 79, 87, 94, 121, 144, 146 (1969). State and local authorities may ban sampling to minors under the 1969 Act, but they may not ban sampling to adults.

Sports Sponsorship. Philip Morris does not sponsor sports contests aimed at juveniles. At the June 8 hearing, some witnesses expressed concern that the Maine Legislature last year rejected a proposed ban on sports

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sponsorship by cigarette manufacturers because of a belief that the ban was preempted by federal law. Contrary to the suggestion of these witnesses, however, the proposed legislation would have banned all sports sponsorship in Maine by cigarette manufacturers -- not just sponsorship of contests involving juvenile athletes and audiences. The proposed legislation also would have banned all sponsorship by cigarette manufacturers of any "community, * * * recreational or civic events under the registered brand names of a tobacco product." In an opinion dated November 12, 1987, the Attorney General of Maine correctly concluded that the proposed legislation would "certainly" undermine the federal objective of a uniform national system for regulating cigarette advertising and promotion, free from "additional" state requirements or prohibitions.

Billboards. Some witnesses at the June 8 hearing also suggested that state and local authorities should have the power to restrict cigarette advertising on billboards located in areas "frequented by children." The concern of these witnesses appears to be that cigarette manufacturers deliberately advertise on billboards directly facing playgrounds or schools. This concern is without foundation. As a matter of policy, Philip Morris and the other members of the industry avoid advertising to audiences that consist primarily of

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children. We are not aware of any Philip Morris advertisements on billboards directly facing playgrounds or schools.

It would be unrealistic for Congress to give state and local authorities the power to determine the location of cigarette billboard advertising, based on their own individual views of how far such advertising should be from centers of youth activity. One community might set the limit at 100 yards, another at 1,000 yards. Still another might prohibit cigarette billboard advertising on any portion of a school bus route. Some communities undoubtedly would attempt to ban all cigarette billboard advertising on the ground that minors are bound to see billboard advertising, wherever located.

The possibility of "diverse" and "nonuniform" treatment of cigarette advertising inherent in such a system is obvious -- as is the likelihood that much cigarette advertising to adults would be banned by state and local authorities, in the name of protecting the health and welfare of children. Such an outcome would violate the basic First Amendment principle that "[t]he Government may not 'reduce the adult population * * * to reading only what is fit for children.'" Bolger v. Youngs Drug Products Corp., 463 U.S. 60, 79 (1983) (Rehnquist, J., concurring). It also would be flatly at

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odds with the premise of the Federal Cigarette Labeling and Advertising Act that cigarette advertising to adults should be allowed, as long as it includes the federally-mandated health warnings.

Mass Transportation. Some witnesses at the June 8 hearing also suggested that state and local authorities should be given the power to "refuse" cigarette advertising in mass transportation facilities because they are publicly owned -- even if the same state and local authorities could not forbid cigarette advertising on privately owned property. The Federal Cigarette Labeling and Advertising Act, of course, does not distinguish between publicly owned and privately owned property in preempting state and local requirements or prohibitions with respect to cigarette advertising or promotion.

Under the First Amendment, the distinction suggested by these witnesses consistently has been rejected as a justification for content-based censorship. Congress should not effectively endorse such censorship. Once public transportation systems have opened their property to commercial advertising generally, advertisements for particular products may not be excluded simply because those in charge do not like their message. At least six federal courts have invalidated restrictions

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on advertising in mass transportation facilities on precisely this ground.

Outright Bans. Congressman Mike Synar (D-Oklahoma), a key swing Democrat on the House Energy and Commerce Committee, authored legislation in the 100th Congress to ban all cigarette advertising and promotion. Several hearings were held on his bill, H.R. 1272.. The national exposure Synar has gained on this issue, combined with Canada's recent enactment of a tobacco ad ban, makes it likely that he will renew his legislative push in 1989.

Proponents of ad bans premise their campaign on two assumptions: first, that cigarette advertising is intended to increase overall cigarette consumption; and second, that the most important effect of cigarette advertising is to create a class of new, young smokers. As a consequence, they argue, banning cigarette ads would reduce smoking in general and by young people in particular. Neither assumption withstands serious empirical scrutiny.

Experts in marketing and consumer behavior have made it clear in their testimony before the Energy and Commerce Committee that cigarette advertising does not convert non-smokers into smokers, encourage current smokers to smoke more or discourage smokers from quitting. Instead, the real influence of cigarette

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advertising is on the particular brand chosen by those who independently choose to smoke. As to young people, the basic conclusion of serious researchers is reflected in the statement of Dr. Mortimer Lipsett, Director of NIH's National Institute of Child Health and Human Development: "the most forceful determinants of smoking are parents, peers and older siblings."

Several countries have banned or severely restricted cigarette advertising, including Italy, Singapore, Norway, Finland, Sweden, Thailand and Taiwan. The evidence from these countries shows that cigarette advertising bans do not affect cigarette consumption trends.

For example, in countries where a tobacco advertising ban has been imposed, either the ban was not followed by a decrease in tobacco consumption, or the ban took place after a decline in smoking had already started. In neither instance was the drop in smoking related to the imposition of the ban.

Moreover, studies comparing smoking by young people in countries where ads are banned and those where ads are allowed confirm that advertising has no significant influence on the decision by young people to start smoking. Indeed, the studies reveal that the incidence of smoking among young people is higher in

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many places where cigarette advertising is banned than where it is not.

The advertising ban legislation also raises serious First Amendment, commercial free-speech issues. Anti-tobacco advocates assert that in light of its recent decision in Posadas v. Tourism Co., 106 S.Ct. 2968 (1986), the Supreme Court would uphold legislation to ban cigarette ads. In contrast, many other groups -- including the American Civil Liberties Union, newspaper and magazine publishers, broadcasters, and eminent legal scholars like Philip Kurland of the University of Chicago and Marvin Redish of Northwestern University -- have made a persuasive case that Posadas was wrongly decided and will not withstand future Supreme Court scrutiny. Whatever the outcome of this formal legal debate, it is clear that the policy assumptions underlying ad ban legislation -- that cigarette advertising raises the overall demand for cigarettes, and influences young people to begin smoking -- are without substantial foundation.

Eliminating Tax Deductions for Advertising. As an alternative to a wholesale ban on cigarette advertising, Senator Bill Bradley (D-NJ) and others have proposed eliminating cigarette companies' ability to deduct advertising expenses for federal corporate income tax purposes. Eliminating the deductibility of .

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advertising is based on the same faulty premise as the broader advertising ban: that cigarette advertising raises overall demand for cigarettes and induces young people to start smoking. This proposal also raises significant First Amendment concerns.

Moreover, elimination of the tax deduction for cigarette advertising would set a dangerous precedent that ought to concern all companies that advertise products and services, as well as the advertising, publishing and broadcast communities as a whole. Clearly, opponents of products such as alcoholic beverages, all terrain vehicles, handguns, and birth control devices would argue that manufacturers of those products do not "deserve" to be able to deduct their advertising expenses. Soon, no company that makes a controversial product or sells a controversial service would be safe from such proposals, and the Congress would be mired in fruitless debate about which advertising expenses are "good enough" to qualify for tax deductions.

Vending Machines and Federal Minimum Smoking Age. The campaign by anti-smoking advocates to prevent smoking by young people is often accompanied by proposals to ban or restrict vending machine sales and to impose a national minimum age for purchasing cigarettes. Neither proposal merits serious consideration by the Congress

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since, to the extent states and local communities perceive any problem, they are taking action in both areas.

While vending machine sales provide important revenue to thousands of groceries, restaurants, and other retailers and businesses, they constitute a relatively small percentage of cigarette sales. Even if vending machines were wiped off the face of the earth it would not solve the problem of cigarette purchases by minors. In addition, many vending machines are located in places -- e.g., bars and nightclubs -- where minors are not allowed to begin with. Other vending machines are located in stores and restaurants where proprietors can easily monitor vending machine purchases. Further, a variety of states -- including Iowa, Utah, Maine, Alaska and Wisconsin -- have recently imposed restrictions on vending machine sales of cigarettes, as have a number of cities and counties. Similar legislation is pending in a variety of other jurisdictions. Thus, where vending machine sales are perceived to be a problem, state and local governments are fashioning solutions tailored to their particular communities.

Moreover, nearly all states impose a minimum age requirement for the purchase of cigarettes. Indeed, 40 states prohibit cigarette sales to persons under 18, and nearly 20 states go further and prohibit possession of

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cigarettes by those under 18. Thus, there is already sufficient law in the area of sales to minors. If there is a problem, what is needed is better enforcement of existing laws. If enforcement is to be effective, it will have to be done by local officials devoting local resources to the enforcement effort. In that context, imposing federal obligations on top of what state and local officials already believe is adequate could lead to resentment and a diminution in the local enforcement effort.

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JUL 3 1988

ATLANTA JOURNAL &
CONSTITUTION

Foes of Smoking Want to Snuff Out Ads for Cigarettes

Their Movement Has Picked Up Steam Since N.J. Jury's Recent Liability Verdict

1-D By Melissa Turner
Staff Writer

The billboard depicts a long-legged, swimsuit-clad woman lolling under a crystal-clear waterfall. No cigarette package. No cigarette. No smoke. Only the slogan, "The Refreshest," and a brilliant green Salem logo.

RJR Nabisco Inc.'s new visual campaign for its menthol cigarette may assuage consumers' growing fears of the dangers of smoking with symbols of vigor and vitality.

That's why anti-smoking activists, including some in Congress, would like to take billboards like that down. They believe that a recent New Jersey jury's verdict finding a cigarette manufacturer partially liable in the death of a lung-cancer victim revives prospects for legislation aimed at further regulating the cigarette industry.

"The jury's decision represents a significant and long-overdue breakthrough," said Rep. Henry A. Waxman (D-Calif.) when the New Jersey verdict was announced. Waxman is chairman of the House subcommittee on Health and the Environment, which has scheduled hearings this month on proposed federal regulation of the sale and promotion of tobacco.

"This crack in the tobacco industry's liability armor will have implications far beyond the specifics of this case," he said.

The bills pending in Congress would have wide-ranging results, from eliminating the tax deductibility of cigarette advertising to restricting smoking on all commercial airline flights, as well as adding addiction warnings on cigarette packaging.

But one bill, put forth by Rep. Mike Synar (D-Okla.), has received the most attention and debate since its introduction two years ago. Synar wants to prohibit all cigarette advertising. Congress banned cigarette advertising on radio and television in 1970, and, since then, many newspapers, including The New York Times, have rejected cigarette ads.

But now Synar, backed by many anti-smoking groups, wants the ads out of magazines, newspapers and sports stadiums, and off race cars and billboards. His bill has gained support with each succeeding session in Congress, and now has at least 30 sponsors.

Even a conservative politician from the tobacco state of South Carolina, Sen. Strom Thurmond, has said he would have no objections to a ban on cigarette advertising — if it were coupled with a ban on alcohol advertising.

A cigarette advertising ban would affect more than tobacco companies. They spent $634 million on advertising last year, with virtually all going to magazines and outdoor advertising. On a worst-case basis a ban could cut profits of some magazine and outdoor advertising firms by 4 percent to 6 percent, according to Diana K. Temple, tobacco analyst with Salomon Brothers Inc.

Ban advocates argue that all advertising should be eliminated because it induces youngsters to smoke, rather than propels smokers to switch brands as the cigarette companies argue.

Although the cigarette companies are fighting proposed bills in the House and Senate, it's not clear that an advertising ban would hurt them.

A number of industry analysts believe a ban would allow the two largest cigarette makers — Philip Morris and RJR — to save $1.5 billion to $2 billion a year between them, and that their sales would only be marginally affected.

And two Clemson University economists suggest an ad ban would be a small bonanza for the industry. In an extensive study, they found that after ads were banned from radio and television in 1970, cigarette company profits increased.

In their study, "Finessing the Political System: The Cigarette Advertising Ban," Mark L. Mitchell and J. Harold Robbins found that the ban boosted profits because it lessened entry to the industry, hence competition, and made the Fairness Doctrine — requiring stations to carry anti-smoking messages as well as cigarette ads — inoperable.

An ad ban would make it almost impossible for an existing brand to take away market share from anoth-

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WILLIAM BERRY/Staff

Construction worker Andrew J. Freeman sits on a MARTA train near a Marlboro advertisement. Foes would limit, if not ban, such promotions, and tobacco firms are fighting their efforts.

5-D **Ads**

From Page 1D

er, and it would be an almost insurmountable barrier to entering the cigarette market, says Larry C. White, author of "The Merchants of Death," an uncomplimentary look at the tobacco industry.

As a result, the current brands would be frozen into their market shares for the foreseeable future he suggests.

"If there were no advertisments' how would consumers have ever found out about low-tar and low-nicotine cigarettes?" asks David Fishel, the chief spokesman for R.J. Reynolds Tobacco Co. "Advertising is the primary way consumers get information about products."

Of course, a ban would make it difficult for RJR to launch its much-ballyhooed smokeless cigarette in test markets later this year. RJR is banking on its "revolutionary" new product to help it close the gap on its longtime rival, Philip Morris, which controls 38 percent of the U.S. tobacco market. RJR has 33 percent.

teenagers, who presumably represent the future of the industry more than current adult smokers.

How young smokers are influenced by advertising was at issue in the New Jersey case. The jury in Newark found that ads run by Liggett Inc. in the 1960s such as one headed "Just What the Doctor Ordered," contained an express warranty of the safety of Liggett's Chesterfield and L&M brands, the brands cancer victim Rose Cipollone started to smoke at 16

co companies' position from advertising companies and the ACLU."

Even Waxman, perhaps the tobacco companies' greatest antagonist in Congress, said last week that his hearings starting July 29 are designed to build support for legislation in the 101st Congress starting in January.

While many analysts believe an excise tax hike is one of the most likely pieces of legislation to pass

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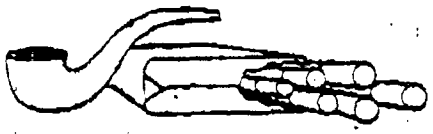

Where tobacco ad dollars go

(1997 spending)

	Advertising for cigarettes, tobacco and accessories	Tobacco's share of all ad spending in each medium
Magazines	$333.58 million	6.2%
Outdoor advertising	$164.76 million	23.4%
Newspapers	$27.02 million	0.4%
Local television	$5.54 million	0.1%
Network radio	$1.65 million	0.3%
Network television	$1.17 million	0.0%
Cable networks	$431,000	0.1%
Total	$534.35 million	2.1%

Source: BAR/LNA Multi-Media Service

...ext year. others disagree. The tobacco companies have become particularly able at mobilizing their constituency," suggests Roy Burry, analyst with Kidder, Peabody.

Philip Morris, for example, launched a multimillion-dollar advertising campaign last week capitalizing on findings of a company-sponsored Roper Organization poll. The first ads, placed last week in 19 daily newspapers across the country, carried the headline "$1 Trillion Is Too Much Financial Power to Ignore."

The figure reflects the combined household income of the 55.8 million American smokers.

Cigarette ad spending, company by company

Millions of dollars per year

	Philip Morris	RJR	Batus	American Brands	Lorillard	Liggett	Industry
1983	$205.1	$242.1	$81.4	$40.2	$78.6	$10.6	$658.0
1984	204.5	283.1	58.5	36.1	72.8	8.6	664.6
1985	234.3	245.8	54.8	34.5	60.0	14.2	643.6
1986	227.7	208.0	37.8	30.6	58.4	3.1	567.7
1987	230.1	158.8	39.4	32.2	46.0	4.0	511.8
1988*	230.0	159.0	40.0	34.0	45.0	4.0	512.0

* Estimate
Source: Salomon Brothers Inc

Rep. Henry A. Waxman

Rep. Mike Synar

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CLEVELAND PLAIN DEALER JUL 3 1988

THE ISSUES/SMOKING

If cigarette advertisements are required to comply with the same standards for truthfulness as other industries, then, and only then, will they be held responsible for false claims.

CPD 7-3 88 2c

By Rep. THOMAS A. LUKEN

The time has come for Congress to declare an objective of a smoke-free society. We will not achieve that goal until the tobacco companies are held legally responsible for their misdeeds.

When a deceased smoker's family sues a tobacco company for wrongful death, they encounter a legal shield of immunity protecting the cigarette company. Congress created that protective armor for the tobacco industry when it passed the Federal Cigarette Labeling and Advertising Act in 1965. Under that law the courts have said that a tobacco company can issue blatantly false and deceptive advertising, and then be absolved of liability by placing a warning label on the cigarette package.

Yet no other industry — not even "smokeless tobacco" products such as snuff and chewing tobacco — enjoys such a shield.

If cigarette advertisements are required to comply with the same standards for truthfulness as other industries (as provided in my bill, the Cigarette Testing and Liability Act of 1988, H. R. 4543), then, and only then, will they be held responsible for false claims in advertising.

Cigarettes are the most dangerous product sold in the United States. The Surgeon General of the United States says that 320,000 Americans die each year from smoking. By comparison, only about 4,000 Americans die each year from cocaine and heroin. In addition to causing these 320,000 deaths each year, smoking-related diseases, according to the United States Office of Technology Assessment, cost $65 billion in extra medical costs and loss of productivity each year.

The absurdity of the current situation for cigarette advertisements is illustrated by the recent trial in New Jersey, where a federal jury awarded Antonio Cipollone $400,000 in damages because of his wife's death in 1984. Rose Cipollone began smoking Liggett & Myers' cigarettes in 1942. In 1968 she switched to Philip Morris brand. In 1974 she began smoking a Lorillard brand. The jury found that her death from lung cancer in 1984 was caused by smoking. However, while the jury found Liggett & Myers was responsible for her death, Philip Morris and Lorillard were held not responsible because she didn't smoke their cigarettes before 1966.

My bill also would make it clear that state and local governments can use their traditional power to protect the health of their citizens by regulating cigarettes in the same way they regulate other products. The need for such granting of authority to the states was highlighted when witnesses at a recent hearing told us that cigarette companies have warned local governments that they cannot pass laws prohibiting the distribution of free cigarette samples. Many state and local governments want to ban free cigarette samples as a means of preventing juveniles from obtaining these harmful products. We also heard testimony that the cigarette companies told the Maine Legislature that under federal law Maine could not restrict advertising of cigarettes in connection with children's skiing events.

If my bill does not pass, it will be a testament to the wealth and power of the tobacco industry. In 1985, the last year for which figures are available, the cigarette industry earned profits of $2.45 billion. In 1986 the cigarette industry spent $2.4 billion advertising and promoting cigarettes. The tobacco companies are reported to have spent $50 million just in defending the New Jersey lawsuit involving Mrs. Cipollone.

Yet, in our system of government the voters are more powerful. I urge you to let your congressman and senators know whether you think the tobacco companies should be held responsible when smokers die after they have been lulled into a false sense of security as a result of false and deceptive cigarette advertising.

Rep. Thomas A. Luken, D-Ohio, is chairman of the House Energy and Commerce subcommittee on transportation, tourism and hazardous materials. This was written for Scripps Howard.

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BURRELLE'S NEWSEXPRESS PAGE 1 OF 1

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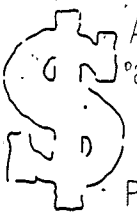
$1 trillion is too much financial power to ignore.



America's 55.8 million smokers are a powerful economic force. If their household income of $1 trillion were a Gross National Product, it would be the third largest in the world. The plain truth is that smokers are one of the most economically powerful groups in this country. They help fuel the engine of the largest economy on the globe.

The American Smoker- an economic force.

PHILIP MORRIS
MAGAZINE

Presented by Philip Morris Magazine in the interest of America's 55.8 million smokers.

Source: The Roper Organization

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Source: https://www.industrydocuments.ucsf.edu/docs/rhbj0000

Today, 21 million American smokers will go out to eat. That's a market you can sink your teeth into!

America's smokers love to go to restaurants. They do so with great frequency. Their tastes in food span the globe— American, Italian, Chinese, French, Japanese and Mexican cuisines. America's smokers feed this country's food service industry.

The American Smoker- an economic force.

PHILIP MORRIS
MAGAZINE

Presented by Philip Morris Magazine in the interest of America's 55.8 million smokers.

Source: The Roper Organization

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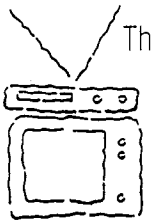
Source: https://www.industrydocuments.ucsf.edu/docs/rhbj0000

American smokers and their families turn on 38 million TVs, 750,000 CD players, and 23 million VCRs. Now that's home entertainment!



These numbers are only part of the whole alphabet soup of home entertainment electronic equipment owned by America's smoker households. They also own more than 4.5 million home computers and 29 million stereos. Those who choose to light up continue to buy millions of TVs, CDs or VCRs each year. America's smokers keep the consumer electronics industry plugged in.

The American Smoker- an economic force.

PHILIP MORRIS
MAGAZINE

Presented by Philip Morris Magazine in the interest of America's 55.8 million smokers.

Source The Roper Organization

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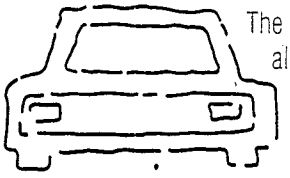
Source: https://www.industrydocuments.ucsf.edu/docs/rhbj0000

Enough cars to stretch bumper-to-bumper around the world four times is too much horsepower to ignore.



The 55.8 million smokers—like all Americans—love cars. In fact, smoker households own more than 35 million cars. Placed bumper-to-bumper, their cars would create a line approximately 100,000 miles long. And more than 5 million of the new automobiles sold last year were purchased by smokers and their families. America's smokers also buy American—fully 83% of their cars were made in the U.S.A. Smokers help keep America on the road to prosperity.

The American Smoker- an economic force.

PHILIP MORRIS
MAGAZINE

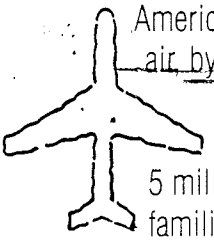
Presented by Philip Morris Magazine in the interest of America's 55.8 million smokers.

Source: The Roper Organization.

2046737557

Source: https://www.industrydocuments.ucsf.edu/docs/rhbj0000

More than 24 million of this country's smokers will travel this summer. Make room, America!



America's smokers travel by land, by air, by sea. They travel far and wide. During the summer, resort hotels alone will welcome 5 million smokers and their families. More than 7.5 million will travel the U.S.A. More than 2 million will head overseas. And 5 million will take to the air. Spring, summer, winter and fall, smokers keep the travel industry of America occupied.

The American Smoker- an economic force.

PHILIP MORRIS
MAGAZINE

Presented by Philip Morris Magazine in the interest of America's 55.8 million smokers.

Source The Roper Organization

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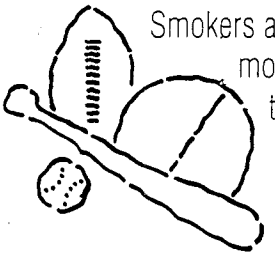
America's smokers attend enough sporting events each year to fill the Astrodome 3200 times.



Smokers are some of America's most avid sports fans! In the past year, 3 million have gone to at least one pro basketball game, 5 million have counted touchdowns at one or more pro football games, and 5.5 million attended at least one pro baseball game. Smokers keep the business of American sports in play.

The American Smoker- an economic force.

PHILIP MORRIS
MAGAZINE

Presented by Philip Morris Magazine in the interest of America's 55.8 million smokers.

Source The Roper Organization

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PRIVILEGED AND CONFIDENTIAL
ATTORNEY WORK PRODUCT

THE SCIENTIFIC DEBATE ON SMOKING AND HEALTH

Over the past thirty years, Congress has held twenty-two sets of hearings on smoking and health. Typically, congressmen and senators, government health and regulatory officials, representatives from the voluntary health organizations, independent scientists, tobacco industry executives, and other interested individuals have all provided testimony for the record.

During these hearings, two consistent lines of questioning have been the state of the scientific evidence associating smoking with various diseases and the research efforts sponsored by the industry to respond to the issues raised by such evidence. Although it is assumed by many of our opponents that the scientific community has long been "unanimous" in concluding that smoking "causes" those diseases, the fact is that a number of respected scientists have testified that the question of whether cigarette smoking causes disease remains unanswered. The passage of time has not diminished the flaws in the case for causation: many of the reasons for skepticism that were cited by scientists in the 1950s and 60s are still valid concerns in the 1980s.

At the same time, it is clear that Philip Morris and the other companies in the industry have behaved

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responsibly in funding a substantial research effort by independent scientists into the wide-ranging issues of "smoking and health." Those efforts -- as described below -- have not always resulted in findings favorable to the industry. Nevertheless, the industry has supported that research -- and those findings have been published -- in order to respond to the issues raised in the scientific debate over smoking and health.

I. THE NATURE OF THE SCIENTIFIC EVIDENCE ON SMOKING AND HEALTH

A. Epidemiological Data

Probably the most important part of our critics' case are the various epidemiological studies associating cigarette smoking with various diseases. A number of independent scientists, however, have expressed concern about the interpretation of such epidemiological data. They were unimpressed by the numbers of such studies, because of concern about the underlying validity of the evidence.

For example, in the early 1980s, Professor Eleanor Macdonald, in criticizing many of these studies said:

> "There is a concept widely accepted by public health schools that if many studies, in themselves not based on definable populations, all arrive at the same conclusion, then that conclusion must be valid. This concept

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> disregards the fact that the same intrinsic error in method might produce the same result." Comprehensive Smoking Prevention Education Act, House Subcommittee on Health and Environment, Appendix ("1982 House Appendix"), p. 683.

A similar view was expressed by Dr. Laurence Kupper during the same hearings:

> "The main prospective (i.e., follow-up) studies concerning smoking and its relationship to mortality and morbidity from certain diseases (as discussed in the latest Surgeon General reports) do not address the issue of causality. The claim that the conclusions reached are 'impressively uniform and consistent' does not mean that a causal relationship has been established, but only that approximately the same observed associations keep appearing. In fact, the biases inherent in these studies preclude the right to claim that causality has been demonstrated. Actually, one could argue that the same associations keep appearing because the same biases are present in each study.
>
> "The fact that so many studies have produced a positive association between smoking and lung cancer has led many people to make the false conclusion that 'quantity means quality.' The truth of the matter is that repeatability (i.e., the ability to produce the same estimated association) does not imply accuracy (i.e., the ability to produce a valid estimate of the true association). In this regard, one study free from all bias and producing a valid measure of the true smoking-lung cancer association is worth more than a thousand biased studies, all of which provide the same distorted estimate of the true association." Id. at 656.

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Specific criticisms of using epidemiological studies to "prove" causation include the following points:

First, epidemiological studies can show a statistical association between factors, but they cannot demonstrate a causal relationship. Even the 1964 Surgeon General's Report conceded this: "Statistical methods cannot establish proof of a causal relationship in an association." (p. 20)

Second, in epidemiological studies smokers, ex-smokers and nonsmokers are not randomly assigned to their study groups. Instead, they make their own decisions about smoking, and they, therefore, constitute "self-selected" samples. Such self-selection can introduce considerable bias into an epidemiological study.

Third, many of the large-scale, prospective studies examining the relationship between smoking and disease did not adequately account for the potential effects of other factors that could affect the results. These factors include general lifestyle, alcohol consumption, occupational and environmental exposures, genetics, aging and the immune processes. As one statistician has commented:

> "In general it is ironic that while studies of environmental or occupational health effects are

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> constrained to take population smoking habits into account, studies on smoking effects seldom bother to search for a report on occupational exposures." Sterling, Cigarette Smoking and Disease, 1976 Hearings Before Senate Subcommittee on Health, p. 456.

In sum, the use of epidemiological data to indict smoking as a "cause" of disease has many flaws, and neither the industry nor independent scientists have acted irresponsibly in noting those problems in the public debate over smoking and health.

B. Specific Evidence Relating Smoking to Various Diseases

A large percentage of the scientific debate has focused on the question of whether cigarette smoking causes lung cancer. From the first (1957) to the most recent (1983) hearings, Congress has heard scientists and physicians express their skepticism about the relationship between smoking and this disease. In 1957, Dr. Joseph Berkson, of the Mayo Clinic, testified that "there is serious doubt as to the scientific validity" of the conclusion that smoking causes lung cancer. False and Misleading Advertising, Hearing Before House Committee on Government Operations, 75. In 1964, Dr. Thomas Burford, Chief of Thoracic and Cardiovascular Surgery at Barnes Hospital in St. Louis, Missouri said "careful study of the literature will reveal that many thoughtful studies by competent people concur in this

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opinion that the cause of lung cancer, or for cancer in any organ is unknown. Without needlessly belaboring the issue it is obvious that there is a large volume of good scientific evidence which tends to refute the premise that cigarette smoking is causally related to cancer of the lung." Cigarette Labeling and Advertising, Hearings Before House Committee on Interstate and Foreign Commerce, 201-02. In 1983, Dr. Walter Booker said "Despite what those in the legislative arena might believe, the cause or causes of cancer of the lung (and other organs) remain unknown. Both smokers and nonsmokers contract cancer and other diseases often associated with cigarette smoking, and we still don't know why." Smoking Prevention Health and Education Act, Hearings Before Senate Committee on Labor and Human Resources, 287.

The belief of these scientists that smoking has not been shown to cause lung cancer rests not only on certain of the generic criticisms of the epidemiological "case" described above, but also on factors specific to that particular disease. For example, ostensibly parallel increases in the 20th century between the consumption of cigarettes and the incidence of lung cancer have been cited as one piece of evidence that smoking causes lung cancer. Yet, both pathologists and thoracic surgeons have repeatedly testified that the

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data on lung cancer trends over time are not dispositive because dramatic improvements in physicians' ability to diagnose lung cancer caused more lung cancers to be reported in official health statistics. As Dr. Rosenblatt said in 1965, "statistics showing a tremendous increase in lung cancer during the past thirty years are misleading. The increase is only apparent and is the result of greater skill in the detection of the disease. All the techniques currently used to diagnose lung cancer were either discovered or perfected during the past three decades. . . . Prior to the development of present diagnostic methods many lung cancers had been erroneously diagnosed as tuberculosis, pneumonia, pleurisy, abscess, or even brain tumor." Cigarette Labeling and Advertising Hearings Before House Committee in Interstate and Foreign Commerce, 248-49.

Similarly, changes in the International Classification of Diseases in the mid-20th century have inappropriately combined data for cancers that originated in the lung with cancers that spread to the lung from another part of the body. Since major internal cancers frequently spread to the lung, these secondary lung cancers have been included in the statistics on smoking and lung cancer. This combination of primary and secondary lung cancers in some statistics has made it impossible accurately to establish a

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statistical relationship between primary lung cancer and any agent, including cigarette smoking.

Another issue that has repeatedly arisen is the proper interpretation of the experiments by Ernst Wynder and other scientists claiming the production of skin cancers after skin painting with tobacco tar.

For several reasons, such skin-painting studies are not directly applicable to the human smoking experience. First, the mice used in the experiments have been specially bred to be extremely sensitive to any possible carcinogenic activity, and they therefore react positively to many substances which are not carcinogenic in humans. For example, in the 1950s, Dr. Jonathan Hartwell of the National Cancer Institute compiled a list of animal carcinogen studies which included such common items as fructose (fruit sugar), glucose (processed sugar), and even lactic acid (made naturally by the human body). He found hat 481 of the 2108 common chemical compounds tested (23%) were "carcinogens" in such animal studies. But, neither Dr. Hartwell nor any other responsible scientist ever concluded that these many "animal carcinogens" were perforce carcinogenic in man.

Second, the substance that is applied to the backs of the mice in such skin-painting experiments -- condensate created by solidifying tobacco smoke and

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mixing it with a chemical solvent -- is very different from the fresh whole smoke that a smoker inhales. It was for that reason that Philip Morris and the other tobacco companies sponsored animal studies by independent scientists which more closely resemble the human smoking condition -- i.e., experiments in which the animals inhale fresh, whole smoke. To this day, the many studies sponsored by Philip Morris have failed to find experimentally produced lung cancer in any of the thousands of laboratory animals subjected to fresh, whole smoke.

Third, the organ involved -- mouse skin -- is obviously quite different from a human lung. Once again, all scientists recognize the importance of inhalation studies which would confirm or, as has been the case, cast doubt on the skin-painting results.

Finally, the doses applied in mouse skin painting are massively greater than the doses of the particulate matter which a smoker would encounter in a normal smoking situation. Once again, the inability to predict human lung cancer from smoking based on such animal work is clear. As Dr. Greene stated at the 1957 hearings, "the [skin-painting] experiments demonstrated that tobacco tar extracted by a special technique induces cancer in the skin of CAF mice, and nothing more. This point is of little significance to workers in the field

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who have found that under certain conditions a multiplicity of substances in every day use will induce cancer in mice of highly suspectible strains."

For these reasons, the industry properly insisted on evidence from inhalation experiments before it would agree that cigarette smoking causes lung cancer (or other pulmonary disease). Over the years, many researchers -- both supported by industry grants and completely independent of any industry connection -- have conducted such experiments where test animals have been forced to take in large doses of whole smoke. One study recently concluded by Microbiological Laboratories (on behalf of the industry) involved thousands of animals in a lifetime experiment.

With a few aberrational exceptions, these studies have not found any significant pathological changes, and none have found credible evidence of cancerous changes.[1] As Arthur Frust, the Director Emeritus of the University

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[1] One of the most thoroughly discussed animal studies was the beagle dog inhalation study conducted by Dr. Auerbach. Dr. Auerbach claimed to find changes which he labeled as cancerous, but his study was severely criticized. Dr. Victor Buhler stated in 1982: "That experiment suffered from severe design defects, and the photomicrographs published with the article would not permit most pathologists to reach the conclusions stated by the authors."

Significantly, Dr. Auerbach was unable to replicate his results in a later study, and his study has never been cited by even such critics as the Surgeon General.

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of San Francisco's Institute of Chemical Biology, said "For many years, I tried to induce lung cancer in animals with cigarette smoke, with no success, despite the most sophisticated smoking machines available. Not only were my colleagues and I unsuccessful, but every other investigator who attempted to induce lung cancer in animals by inhalation of fresh smoke also failed." In summary, he said: "I have concluded that no reliable, reproducible animal studies have shown that the inhalation of cigarette smoke causes lung cancer." Furst, 1983 Senate, pp. 465, 467.

In sum, the totality of the evidence fails to establish that smoking is a cause of lung cancer.

Independent scientists have similarly expressed skepticism about the alleged causal link between smoking and heart disease. They have noted the inconsistent statistical correlations between smoking and heart disease in various populations; the strong genetic influence in heart disease; and the failure of the critics of smoking to establish a mechanism to explain how tobacco smoke might cause heart disease. In 1964, Dr. Henry Russek testified before Congress that "statistical findings linking the tobacco habit to coronary disease afford no proof of a causal relationship" and that his research had shown that emotional stress was far more significant than cigarette

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smoking in the development of heart attacks. Russek, 1964, p. 218. In 1982, Dr. Carl Seltzer, a Fellow of the American Heart Association's Council on Epidemiology, similarly said that "the bill's proposed warning, 'cigarette smoking is a major cause of heart disease,' is not scientifically valid." Seltzer, 1982 House Appendix, p. 757.

In much the same way, the failure of experiments to produce emphysema in animals that have inhaled cigarette smoke casts serious doubt on the statistical associations in various epidemiological studies. At the 1982 House hearings, Dr. Domingo Aviado described his efforts over many years to induce emphysema in laboratory animals exposed to cigarette smoke. None of the experiments produced emphysema in the animals. Dr. Aviado therefore questioned "the logic of finding cigarette smoke the major cause of pulmonary emphysema when primary air pollutants have been shown to cause pulmonary emphysema in experimental animals, and with the same models, cigarette smoking has not." 1982 House Appendix, p. 540.

C. Risk Factor: The Issue and Analysis

The antismoking forces, including the Surgeon General, claim that tobacco kills 350,000 persons per year. The claim is now so commonplace that most of the people repeating it have no idea where it came from or

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what it really means. In fact, the number and the entire computation are subject to serious scientific challenge.

Where did the number come from? A logical inference is that the Surgeon General makes an annual check of smoker medical records (e.g., death certificates) and determines, on some consistent medical basis, the number of cancer or heart disease deaths due to smoking. The reality is quite different. The most recent authority for the 350,000 number is a study published by the Office of Technology Assessment of the U.S. Congress (the "OTA") in September, 1985. That study, like others before it, utilized the following procedure:

(1) The underlying data are not derived from contemporary medical records for smokers (or anybody else), but rather, from an epidemiological study run by the American Cancer Society ("ACS") in the early 1960's.

(2) Those data are used to determine the mortality rates for ACS-nonsmokers for three major diseases: cancer (lung, lip, oral, thorax, esophagus, pancreas and bladder), cardiovascular disease and emphysema/bronchitis.

(3) Those rates are projected on the entire 1985 population to determine the "expected" number of deaths at ACS-nonsmoker rates.

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(4) The expected deaths are subtracted from the actual 1985 deaths and the difference is declared to be "due to smoking." Specifically, the breakdown of extra deaths is as follows:

Cancer	139,000
Cardiovascular disease	123,000
Emphysema/Bronchitis	52,000
Total	314,000

What's wrong with this technique? First, the OTA study assumes that causation is fully established for lung cancer and for a variety of other cancers and heart disease as well; second, it also assumes that the ACS sample is not only representative of the entire United States population, but can be projected forward twenty years; and third, it assumes that smoking is the sole and only cause of "extra" smoker deaths -- an obvious fallacy which permits us to characterize the 350,000 number as essentially meaningless. Each of the "errors" is discussed briefly below.

1. Causation

The 350,000 number has been asserted for so long by so many people that it has become an independent indictment of tobacco. In fact, as described above, it does not represent separate evidence. It is nothing more than an extrapolation of the same "old" epidemiological statistics which are supposed to prove

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causation. The industry has long asserted (correctly) that statistics cannot and do not prove causation. Thus, the first-line response to the 350,000 is clear: "It is not proven."

2. Sampling

Even assuming causation, the studies also postulate that the ACS data are transferrable to the United States population. That is wrong. It is well known that the ACS cohort was considerably healthier than the general population. (They were also more white collar, more white, more suburban, and less occupationally exposed). This is not a small matter. When the "healthier" ACS death rates are projected, there are inevitably going to be "extra deaths." Nonetheless, the whole difference is blamed on smoking.

The sampling problem is further complicated by the time leap from 1965 to 1985. It is one thing to project 1965 ACS death rates on the 1965 population; it is quite another to project those rates 20 years into the future. We are talking about a vastly different group of people in terms of environmental influences, health practices (e.g., exercise, diet), medical treatment, and smoking habits.

Again, this is no small matter. For instance, we know that heart disease rates dropped dramatically in the last twenty years. As a result, had the ACS simply

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projected nonsmoker rates from 1965 to 1985, they would have come up with a big surprise: there would be no extra deaths to blame on smoking. On the contrary, the 1985 population had fewer deaths than "expected". The logical inference, applying the OTA methodology, would be that smoking was protecting people against heart disease. To avoid that result, the OTA postulated that the reduction in heart disease must be due to diminished smoking and adjusted the computation accordingly.

If these sampling problems stood alone, it might be possible to come up with an alternative computation. But the additional issue of "confounding factors" permits us to argue that there really is no calculable number.

3. Confounding Factors

No one denies that cancer and, most particularly, heart disease are multi-factorial diseases. Nor can anyone deny that smokers, as a class, are more exposed to most of these "other factors." This is not to say that smokers have a genetic predisposition to disease. What we are talking about here is life style, the same kinds of choices or personality characteristics or environmental influences that lead people to smoke.

Just last month, the Public Health Service ("PHS") (a copy of which is included in these materials) issued a report seeking an answer to the question

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"whether smokers have other unhealthy habits that may . . . increase their probability of succumbing to serious illness, disability and even death." The report concluded that "smoking is related to other unhealthy behaviors." Included in the survey were hours of sleep (31% of heavy smokers got less than six hours, compared to 21% of never smokers); breakfast habits (an astounding 48% of heavy smokers never eat breakfast, compared to 18% of never smokers); exercise (65% of heavy smokers were "sedentary," compared to 55% of never smokers); and alcohol consumption (22.5% of heavy smokers averaged two drinks a day, compared to 3.6% of nonsmokers). The big surprise was over-eating, where it was expected that smoking would have a uniformly favorable effect. In fact, it turned out that male heavy smokers snack more and are not significantly different in weight.

The PHS results are more notable for their currency and source than for their substance. Had the PHS surveyed the existing literature, it would have found evidence for smoker exposure to a wide variety of "other" risks: socioeconomic factors (blue collar, education, income); occupational factors (job exposures to carcinogens); geographical factors (urban versus rural); other environmental factors (water and air pollution); alcohol; coffee; other dietary habits

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(Vitamin A); drug use; exercise; Type-A personality (aggressive or ambitious); stressed personality (depressed or anxious); situational stress (unemployment and divorce); lack of preventive health care; suicide, accidents, violence and overall risk taking.

In this light, the gross error in the OTA study is that it is "univariate." It takes into account only one health behavior, whereas there are many associated with smoking that might influence mortality. One way to demonstrate this error is to take another health behavior, apply a univariate analysis, and see where it leads. For instance, a recent preliminary report from the University of Minnesota (which Dr. Schuman tried very hard at the Cipollone trial to disown) finds that heavy coffee drinkers have a relative risk for lung cancer of about 7 to 1 (compared to 10-1 for smokers). If we assume that the only important variable is coffee and that a substantial portion of heavy coffee drinkers are smokers, we could do a calculation that re-attributes a very large portion of lung cancer deaths to coffee (from smoking). We could do the same sort of calculation with respect to exercise or stress or Type-A behavior or any of the other factors mentioned above -- if we had the data.

Of course, the result of each of these univariate analyses would be wrong. Each would fail to take into

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account the "confounding" effects of all the others. What is needed is a "multivariate" approach.

In fact, the ACS study did include a lot of the relevant behavioral data. Nonetheless, the ACS and the OTA chose to focus only on smoking. In the Cipollone pretrial discovery, we obtained (for the first time) all the underlying ACS data. Statistician Irwin Miller put that data (for women and lung cancer) into a computer and did a "multivariate" analysis -- i.e., the computer juggled all the health related behaviors simultaneously to see which ones were related to lung cancer. Dr. Miller's conclusion was that a great portion of the apparent relationship between smoking and lung cancer disappeared when other factors were taken into account. He is doubtful, given the interdependence of all the health behaviors and the absence of full data, that any clear multivariate conclusion can be drawn.

At trial, plaintiff offered no rebuttal witness and no real answer. Thus, for now, Dr. Miller's conclusion represents the industry's optimal response to the 350,000.

II. THE INDUSTRY RESPONSE

Notwithstanding the indictment by its critics, the tobacco industry responded in an appropriate fashion to health issues raised about smoking by funding a

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massive, independent research effort. That program has involved hundreds of outside institutions and thousands of independent scientists who have added markedly to science's understanding of a number of diseases and any possible link between them and smoking.

A. Council for Tobacco Research

In 1954, the chief officers of nine tobacco products manufacturing companies in the United States, and five organizations of growers of leaf tobacco and operators of tobacco warehouses formed the Tobacco Industry Research Committee (TIRC). The purpose of the TIRC was to investigate the connection between tobacco use and human health. Although the name, TIRC, was changed in 1964 to the Council for Tobacco Research (CTR), the organization continues to pursue the goals that were established by its founders in 1954.

Seeking to promote objective, independent research on smoking and health, the TIRC formed a group of independent scientists called the Scientific Advisory Board (SAB). This group is composed of highly-regarded scientists who come from well-known universities, laboratories, and medical centers throughout the country. The SAB decides what research will be funded.

CTR has been fortunate in that it has been guided by independent scientists of the highest reputation. Some of the members of the current SAB are: Richard J.

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Bing, M.D., Professor of Medicine, University of Southern California School of Medicine; Roswell K. Boutwell, Ph.D., Professor of Oncology, University of Wisconsin; Michael J. Brennan, M.D., President and Medical Director, Michigan Cancer Foundation; Joseph D. Feldman, M.D., Editor, Journal of Immunology; Leon O. Jacobson, M.D., Professor of the Department of Medicine, University of Chicago; Manfred L. Karnovsky, Ph.D., Harold T. While Professor of Biological Chemistry, Harvard Medical School; Alfred G. Knudson, Jr., M.D., Ph.D., Fox Chase Cancer Center, Institute for Cancer Research; Henry T. Lynch, M.D., Professor and Chairman, Department of Preventive Medicine and Public Health, President, Hereditary Cancer Institute, Creighton University School of Medicine; G. Barry Pierce, M.D., American Cancer Society Centennial Research Professor, University of Colorado Health Sciences Center; Gordon H. Sato, Ph.D., Director, W. Alton Jones Cell Science Center; Sheldon C. Sommers, M.D., Clinical Professor of Pathology, Columbia University; Peter K. Vogt, Ph.D., Chairman, Department of Microbiology, University of Southern California School of Medicine.

When an application reaches CTR, the scientific staff assigns a subcommittee of the SAB to each application. At the SAB meeting, each application is discussed. A member of the subcommittee assigned to the

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application is asked to present a critique of the proposal. Other critiques are read and a general discussion is held regarding the application. A vote is then taken as to whether to approve the application. If the application is approved, each SAB member rates the application as to its relevance to smoking and health. The Scientific Director must follow the advice of the SAB giving preference to those applications given high ratings.

The administrative work of CTR is carried out by a permanent scientific and administrative staff located in New York City. CTR has always had distinguished researchers who have served as its Scientific Director. The first was Dr. Clarence Cook Little, a prominent researcher in the field of genetics. Others who have served in this role are Dr. William U. Gardner, a former Professor of Anatomy at the Yale University School for Medicine, and Dr. Sheldon C. Sommers, a former Professor of Pathology at Columbia University College of Physicians and Surgeons. The present Scientific Director is James F. Glenn, M.D., former Dean of the Mount Sinai School of Medicine. The Scientific Director of CTR is responsible for administering funds for medical and laboratory research. The funds are appropriated by CTR only upon the recommendation of the SAB.

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The Industry Technical Committee (ITC), composed of the research directors of the member companies, acts as an advisory panel for the Scientific Director and the SAB. They provide technical information from industry sources about cigarettes and tobacco. A representative of the ITC may attend SAB meetings, but has no vote on research grants.

The overall administration of CTR has been the responsibility of the Board of Directors, whose members are designated executives of the companies that belong to CTR. The Board of Directors is responsible for administrative decisions such as determining the budget, voting on the by-laws and selecting office space. But the directors have no control over the research decisions of the SAB.

CTR does not operate its own research facility. Rather, research support has been achieved primarily through a program of grants-in-aid, supplemental by contracts with institutions and laboratories. As of 1986, grants and contracts have been approved totaling more than $110,000,000. Grants have been awarded to 592 scientists in 296 medical schools, hospitals and research institutions. Much of this research was jointly funded by non-tobacco interests such as U.S. Public Health Service, the National Institutes of Health, and the National Cancer Institute.

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CTR grantees have made significant contributions to scientific research. They have published over 3,000 papers and articles. They have presented their findings at scientific and medical society meetings too numerous to list. A significant amount of this research has resulted in findings which were contrary to the industry's position on smoking and health. Nevertheless, these adverse findings by those independent scientists were published and have since been cited by critics of the industry such as the Surgeon General.

CTR's efforts to stimulate scientific research extend beyond its grant and contract programs. The Council for Tobacco Research has also cooperated with other agencies in the planning and support of public symposia. For example, CTR has sponsored a large number of informal conferences. For many years, CTR annually made available to every medical school in the United States a fellowship for assisting at least one degree candidate, permitting that individual to spend a summer or other "off" period in research under the direction of a senior staff member.

Since the beginning, CTR has supported research that would identify and define factors that may be involved in the causation of certain chronic diseases associated with cigarette smoking. Approved research

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has covered a wide range of human diseases including many types of cancer, respiratory and cardiovascular diseases. CTR also approved investigation into other medical, chemical, biochemical and pharmacological areas. In addition, CTR has sponsored the study and development of basic laboratory and research techniques and has underwritten reviews of research literature in specific areas.

Research funded by CTR has added much to our present knowledge about cigarette smoking and health. For example, the research has: (1) tested the hypothesis that cigarette smoke acts as a direct contact carcinogen; (2) identified smoke components that affect cilia; (3) investigated whether smoking or nicotine causes cardiovascular disease; (4) attempted to produce pulmonary squamous cell carcinoma in animal inhalation studies. These studies and other published research demonstrate CTR's commitment to examining the role of cigarette smoking in disease causation.

B. Institutional Research

For the past 34 years, Philip Morris has also demonstrated its interest in smoking and health issues by supporting research efforts at other institutions with nonrestrictive funding. Most notably, three separate multi-million dollar projects have been sponsored at three universities: Harvard University,

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the University of California at Los Angeles and Washington University.

A unique and important aspect of this industry grant research is the flexibility in the utilization of the funds. This flexibility has made it possible to provide funding for the initiation of research programs which are then supported by grants from the National Institutes of Health and other agencies as well as providing certain items of core equipment that are shared by the investigators.

Harvard

In September 1972, Harvard Medical School was awarded a broad-based institutional grant by Philip Morris and the other tobacco companies to launch a five-year investigation into pulmonary and cardiovascular diseases. In 1977, the major tobacco manufacturers renewed the grant for an additional three-year period.

The project was under the direction of Dr. Gary Huber, Assistant Professor of Medicine and Director of the Respiratory Diseases Clinic at Boston City Hospital's Channing Laboratory. At the time the project was launched, Dr. Huber was already directing a well-established research program for the study of host defenses in the lung. With the aid of the grant, Dr. Huber was able to expand his program to include

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studies on the relationship between tobacco smoke and health, on differences in host susceptibility to disease, and the effect of other environmental influences, such as air pollutants, on the lung.

Dr. Huber's research project was designed to contain four phases. First, he intended to redirect his existing program toward the study of possible biological effects of cigarette smoke on the lung with the objective of determining what role, if any, cigarette smoking might play in the development of pulmonary disease. Second, he sought to expand existing resources to include more comprehensive studies in the areas of biochemistry, pathology and epidemiology. Third, Dr. Huber planned to develop resources for the study of potential relationships between cigarette smoke and lung cancer. Fourth, he intended to study possible relationships between cigarette smoke and cardiovascular diseases.

During the eight-year period in which the industry sponsored the project, Dr. Huber and his co-workers published approximately ninety articles on their work. In November 1977, Dr. Huber reported that his team had found reductions in the walls of the lung alveoli in their rat population and a modest decrease in the lung surface area. This published report was interpreted by some of our opponents as demonstrating a

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direct link between cigarette smoke exposure and lung changes characteristic of emphysema in experimental animals. Yet, in his presentation, Dr. Huber cautioned that additional research would be necessary to determine if the alterations were analogous to emphysema in humans.

The Harvard grant also made it possible for Dr. Huber and his group to obtain government funding for other smoking-related projects. With funds received from the National Cancer Institute and the National Institutes of Health, he was able to develop a method of monitoring respiration and smoke inhalation patterns of human smokers.

Unfortunately, Dr. Huber was unable to complete the project because of difficulties with the animal inhalation facility used by his group. In 1977, the animal facility was closed on the advice of Harvard veterinarians. A large number of the animals had developed infections. Although Dr. Huber spent many months attempting to find other facilities and the sponsoring companies were making plans for long-term support, a practical solution could not be found. Consequently, Dr. Huber finished pending research, and the program was terminated in June 1980.

UCLA

The commitment of Philip Morris and the other cigarette companies to broad-based research is also

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demonstrated by its support of a biomedical research project from 1974 to 1982 at the UCLA School of Medicine. The UCLA project, under the direction of Dr. Martin J. Cline, Chief of the School of Medicine's Division of Medical Oncology and Hematology, facilitated the expansion of the Division and supplemental ongoing research in three major areas, including the investigation of a possible relationship between tobacco usage and disease, particularly lung cancer as well as exploration of methods for early cancer detection and therapeutic techniques involving immunotherapy and chemotherapy.

The project researchers determined that the ability to define alveolar macrophage function could aid in the analysis of environmental factors and diseases that might influence macrophage function. They could not identify any significant abnormalities or any adverse effect of smoking on the performance of human alveolar macrophages.

The UCLA researchers collaborated with others in an attempt to determine whether the activation of cancer genes is important in causation of tumors. Preliminary data suggested that these genes may be important in the initiation and maintenance of certain tumors.

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The UCLA project also provided new information in the treatment of refractory malignant disease. The researchers in the project were among the first to develop the concept of freezing bone marrow for long-term storage in patients with malignant disease.

As this brief summary suggests, physicians and scientists involved in the UCLA project were able to address fundamental problems of human health and disease. Their efforts have resulted in the publication of numerous articles in scientific and medical journals, in which the support of the participating tobacco companies was acknowledged.

Washington University

The third of the broad-based institutional grants was awarded in March 1971, to Washington University in St. Louis for a basic research program on the immunologic properties of cancer. The project is still being funded today by Philip Morris and three other tobacco companies.

Initially, the project was conducted under the direction of Dr. Lauren Ackerman, an international authority on cancer, and Dr. Paul E. Lacy, head of the University's Department of Pathology and one of the world's leading experts on cell structure. The initial purpose of the program was to find and analyze "foreign" substances or antigens within the cancer cell and to

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utilize those antigens for the early detection, treatment and possible prevention of cancer.

This focus eventually led to immunology studies on the role of antibodies in the growth and development of neoplastic cells; the cellular events involved in antibody production; the morphologic and biochemical structure of the cell membrane; the effect of tumor viruses on the genetic information in the cell; and the effect of physical and chemical carcinogenic agents on the genetic information in cells.

The research findings from this program, reported in more that 300 publications, have made significant contributions to ongoing work aimed at understanding the basic cause, development and treatment of cancer and other diseases. The understanding of the immune system made possible by this research is relevant not only to cancer, but it can also be expected to deliver insights into diseases such as lung emphysema, rheumatoid arthritis, lupus and diabetes.

C. AMA-ERF

The American Medical Association Education and Research Foundation Project for Research on Tobacco and Health (AMA-ERF) was funded by six cigarette manufacturers from 1964 through 1971 with contributions totaling over $15 million. All administrative, scientific and policy decisions, screening of grant

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applications, and distribution of funds were the responsibility of the AMA-ERF Board of Directors and the scientific advisory committee appointed by the board. Other than funding, the project was entirely independent of the tobacco industry.

In 1963, the AMA house of Delegates adopted a recommendation from its Board of Trustees that the AMA-ERF undertake a comprehensive research program on tobacco and health. The AMA-ERF was an existing organization within the AMA, primarily responsible for supporting medical education and fostering scientific and medical research.

An AMA announcement indicated that the project would be financed by a contribution from the AMA and by solicited contributions. Only unrestricted contributions would be accepted. The tobacco industry wrote the AMA early in 1964, offering to contribute $10 million to the project over a five-year period. The industry was responding to the Surgeon General's suggestion that more research was needed. The contributions were made with the understanding that the money would be used only for research on smoking and health.

The AMA-ERF Board of Directors appointed a committee of distinguished scientists, the Committee for Research on Tobacco and Health (CRTH), to develop

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guidelines on research policies and procedures, to identify significant areas of research, and to screen applications for research grants. Three of the original members of the AMA-ERF committee had also served on the Surgeon General's Advisory Committee that produced the 1964 Surgeon General's Report.

Grantees were required to publish their findings in the medical or scientific journal of their choice so that the information would become public through the scientific media. Grantees were also expected to participate in workshops convened by the AMA-ERF committee in 1966, 1970 and 1972 for the purposes of presenting oral reports on their research projects, stimulating an exchange of ideas, and delineating new areas for investigation.

In 1968, during the AMA's Annual Meeting in San Francisco, a presentation was made by some of the grantees to report on their progress. At that time, the AMA-ERF committee indicated that more research was still needed to clarify many of the issues surrounding tobacco and health, including questions of causation. This statement became controversial because at the time it was interpreted as casting doubt on the Surgeon General's 1964 conclusions. The AMA-ERF committee then clarified its position in a statement published in JAMA two months after the annual meeting, affirming that the

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research completed under this project in no way challenged the conclusions of the 1964 Surgeon General's Report.

In August 1971, seven years after the project had begun, the tobacco companies and AMA began to consider phasing out the project. By July 1972, they had agreed to conclude the project. There had been an on-going disagreement between the contribution companies and the AMA-ERF committee about the relevance of some of the research to the question of smoking and health. Some contributing scientists had questioned the relevance of some of the research. The AMA, however, had maintained that all of the research was relevant. In addition, the sponsoring companies felt that, although the original grant-based project had been satisfactory when it was set up on 1964, research directed at specific questions was now needed.

It should also be pointed out that during the entire project, the critics of smoking had become more vocal. The AMA was sensitive to criticism of its involvement in this project. In fact, as early as 1969, a committee of the AMA House of Delegates had considered not renewing the project; however, at that time, it was decided that the project was worthwhile, despite unfavorable public relations consequences.

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The findings of the AMA-ERF project were well publicized both in the medical and scientific literature and in a final summary report by the AMA-ERF committee, produced in a 1978 under the title Tobacco and Health. The information in the final summary was not new since the research results had been published by the grantees in scientific journals over the previous fourteen years. According to the summary, the foundation had sponsored 219 separate research projects that resulted in 795 publications and reports. Copies of that were provided free of charge to institutional members of the Medical Library Association. The AMA also sent copies to those who wrote to request one.

In sum, the AMA-ERF project represents a substantial effort on the part of the tobacco industry to sponsor broad-based research on smoking and health. Disagreement between the company contributors and the AMA about the relevance of some of the research reflects differing points of view and the independent nature of the project.

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The Health Consequences Of Smoking

CANCER

a report of the Surgeon General

U.S. DEPARTMENT OF HEALTH AND HUMAN SE
Public Health Service
Office on Smoking and Health
Rockville, Maryland 20857

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THE SECRETARY OF HEALTH AND HUMAN SERVICES
WASHINGTON, D.C. 20201

FEB 17 1982

The Honorable Thomas P. O'Neill, Jr.
Speaker of the House of Representatives
Washington, D.C. 20515

Dear Mr. Speaker:

I hereby submit to you the 1982 Report on the Health Consequences of Smoking, prepared in accordance with the Public Health Cigarette Smoking Act of 1969 and its predecessor, the Federal Cigarette Labeling and Advertising Act. This is the first report in the series to focus on a single disease entity--cancer.

Scientists inside and outside of Government have evaluated the evidence presented in this report. It joins this Department's previous reports on smoking and health in making publicly available information about one of the major health risks of smoking. These reports reflect the important responsibility of Government to inform its citizens in order that they can make a considered decision about whether to smoke.

Sincerely,

Richard S. Schweiker
Secretary

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PART I. INTRODUCTION AND CONCLUSIONS

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Introduction

Development and Organization of the 1982 Report

The content of this Report is the work of numerous scientists within the Department of Health and Human Services, as well as scientific experts outside the organization. Individual manuscripts were reviewed by experts, both outside and within the Public Health Service, and the entire Report was reviewed by a broad-based panel of 12 distinguished scientists. Many of these scientists are, or have been, directly involved in research on the health effects of smoking. The 1982 Report consists of a Preface by the Surgeon General, a Foreword by the Assistant Secretary for Health of the Department of Health and Human Services, and five Parts, as follows:

- Part I. Introduction and Conclusions
- Part II. Biomedical Evidence for Determining Causality
- Part III. Mechanisms of Carcinogenesis
- Part IV. Involuntary Smoking and Lung Cancer
- Part V. Cessation of Smoking

Historical Perspective

Tobacco use was associated with the possible development of cancer as early as 1761. According to one medical historian, Dr. John Hill (1716?-1775) should be credited with the first report documenting an association between tobacco use and cancer for his work *Cautions Against the Immoderate Use of Snuff*. Hill reported on two case histories and observed that "snuff is able to produce...swellings and excrescences" in the nose, and he believed these to be cancerous. Others credit Soemmerring in 1795 for noting a relationship between cancer of the lip and tobacco use.

It was not until the 1920s and 1930s that investigators began to examine scientifically the possible association of smoking and cancer. In 1928, Lombard and Doering, in the United States, found an association between heavy smoking and cancer in general. Muller and Schairer (Germany) in 1939 and 1944 respectively, and Porter (USA) in 1945, and others, noted higher percentages of smokers among lung cancer patients than among controls. The first major developments in the modern history of investigation of the effects of smoking on health occurred in 1950 with the publication of four retrospective studies on smoking habits of lung cancer patients and controls in the United States by Schrek et al., Mills and Porter, Levin et al., and Wynder and Graham. Each of these noted a consistent, statistically significant association between smoking and cancer of the lung. Other investigators proceeded to further examine the relationship by initiating prospective studies in which large numbers of healthy persons were followed over time and their subsequent mortality noted.

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The first major prospective study encompassing total and cause-specific mortality was initiated in October 1951 by Doll and Hill in the United Kingdom among 40,000 British physicians. Hammond and Horn followed 188,000 males beginning in January 1952 in the United States. These and subsequent prospective studies conducted in the United States, Sweden, Canada, and Japan, found not only that smokers have substantially elevated cancer mortality rates, but also that smokers experience significantly elevated overall death rates.

Cancer has been the second ranking cause of death in the United States since 1937. Provisional vital statistics data for 1980 indicate cancer accounted for almost 21 percent of all deaths in the United States. This compares to 17 percent of all deaths in 1970 and 14.5 percent of all deaths in 1950. Various investigators have suggested that 22 to 38 percent of these deaths can be attributed to smoking, and therefore, are potentially "avoidable" if smoking did not exist as a human behavior. Since 1950, the age-adjusted overall cancer death rate has changed little, whereas the lung cancer death rate has increased dramatically for both males and females.

The male age-adjusted lung cancer rate increased 192 percent during the period 1950–1952 thru 1976–1978. Female lung cancer death rates during this same period increased even more: 263 percent. Since the 1950s, lung cancer has been the leading cause of cancer death among males in the United States, and if present trends continue, will become the leading cause of cancer death in females during this decade; the age-adjusted female lung cancer death rate is projected to possibly surpass the death rate for breast cancer next year. Today, deaths from cancer of the lung represent fully one quarter of all deaths due to cancer in the United States.

In 1962, the year when the Surgeon General's Advisory Committee on Smoking and Health began deliberating the evidence presented in its landmark report, slightly more than 41,000 persons died of lung cancer annually, compared to 18,300 lung cancer deaths in 1950. In 1982, the American Cancer Society estimates 111,000 Americans will die of lung cancer, nearly a three-fold increase in the number of deaths in a 20-year time span.

The Advisory Committee's Report of 1964 judged the causal significance of the association of cigarette smoking and disease by rigid criteria, no one of which alone was sufficient for a causal judgment. The epidemiologic criteria included:

a. The consistency of the association
b. The strength of the association
c. The specificity of the association
d. The temporal relationship of the association, and
e. The coherence of the association

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Corroboration was also sought from other sources, such as clinical autopsy and experimental evidence.

Significant additional scientific evidence linking smoking to cancer, as well as to other tobacco-related diseases, has accumulated since the issuance of that Advisory Committee's Report in 1964. Much of this has been collected, reviewed, and published in annual reports by the Department of Health and Human Services.

The purpose of this Report is to review in depth the many sources of scientific evidence relating cigarette smoking to each cancer by anatomic site, and to evaluate this evidence by the same criteria first established by the Advisory Committee in its 1964 Report, including experimental carcinogenesis and human epidemiologic studies.

Conclusions of the 1982 Report

Overall Cancer Mortality

1. Cigarette smokers have overall mortality rates substantially greater than those of nonsmokers. Overall cancer death rates of male smokers are approximately double those of nonsmokers; overall cancer death rates of female smokers are approximately 30 percent higher than nonsmokers, and are increasing.
2. Overall cancer mortality rates among smokers are dose-related as measured by the number of cigarettes smoked per day. Heavy smokers (over one pack per day) have more than three times the overall cancer death rate of nonsmokers.
3. With increasing duration of smoking cessation, overall cancer death rates decline, approaching the death rate of nonsmokers.

Site-Specific Cancer Mortality

Lung Cancer

1. Cigarette smoking is the major cause of lung cancer in the United States.
2. Lung cancer mortality increases with increasing dosage of smoke exposure (as measured by the number of cigarettes smoked daily, the duration of smoking, and inhalation patterns) and is inversely related to age of initiation. Smokers who consume two or more packs of cigarettes daily have lung cancer mortality rates 15 to 25 times greater than nonsmokers.
3. Cigar and pipe smoking are also causal factors for lung cancer. However, the majority of lung cancer mortality in the United States is due to cigarette smoking.
4. Cessation of smoking reduces the risk of lung cancer mortality compared to that of the continuing smoker. Former smokers who have quit 15 or more years have lung cancer mortality rates only slightly above those for nonsmokers (about two times

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greater). The residual risk of developing lung cancer is directly proportional to overall life-time exposure to cigarette smoke.

5. Filtered lower tar cigarette smokers have a lower lung cancer risk compared to nonfiltered, higher tar cigarette smokers. However, the risk for these smokers is still substantially elevated above the risk of nonsmokers.
6. Since the early 1950s, lung cancer has been the leading cause of cancer death among males in the United States. Among females, the lung cancer death rate is accelerating and will likely surpass that of breast cancer in the 1980s.
7. The economic impact of lung cancer to the nation is considerable. It is estimated that in 1975, lung cancer cost $3.8 billion in lost earnings, $379.5 million in short-term hospital costs, and $78 million in physician fees.
8. Lung cancer is largely a preventable disease. It is estimated that 85 percent of lung cancer mortality could have been avoided if individuals never took up smoking. Furthermore, substantial reductions in the number of deaths from lung cancer could be achieved if a major portion of the smoking population (particularly young persons) could be persuaded not to smoke.

Laryngeal Cancer

9. Cigarette smoking is the major cause of laryngeal cancer in the United States. Cigar and pipe smokers experience a risk for laryngeal cancer similar to that of a cigarette smoker.
10. The risk of developing laryngeal cancer increases with increased exposure as measured by the number of cigarettes smoked daily as well as other dose measurements. Heavy smokers have laryngeal cancer mortality risks 20 to 30 times greater than nonsmokers.
11. Cessation of smoking reduces the risk of laryngeal cancer mortality compared to that of the continuing smoker. The longer a former smoker is off cigarettes the lower the risk.
12. Smokers who use filtered lower tar cigarettes have lower laryngeal cancer risks than those who use unfiltered higher tar cigarettes.
13. The use of alcohol in combination with cigarette smoking appears to act synergistically to greatly increase the risk for cancer of the larynx.

Oral Cancer

14. Cigarette smoking is a major cause of cancers of the oral cavity in the United States. Individuals who smoke pipes or cigars

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experience a risk for oral cancer similar to that of the cigarette smoker.

15. Mortality ratios for oral cancer increase with the number of cigarettes smoked daily and diminish with cessation of smoking.
16. Cigarette smoking and alcohol use act synergistically to increase the risk of oral cavity cancers.
17. Long term use of snuff appears to be a factor in the development of cancers of the oral cavity, particularly cancers of the cheek and gum.

Esophageal Cancer

18. Cigarette smoking is a major cause of esophageal cancer in the United States. Cigar and pipe smokers experience a risk of esophageal cancer similar to that of cigarette smokers.
19. The risk of esophageal cancer increases with increased smoke exposure, as measured by the number of cigarettes smoked daily, and is diminished by discontinuing the habit.
20. The use of alcohol in combination with smoking acts synergistically to greatly increase the risk for esophageal cancer mortality.

Bladder Cancer

21. Cigarette smoking is a contributory factor in the development of bladder cancer in the United States. This relationship is not as strong as that noted for the association between smoking and cancers of the lung, larynx, oral cavity, and esophagus. The term "contributory factor" by no means excludes the possibility of a causal role for smoking in cancers of this site.

Kidney Cancer

22. Cigarette smoking is a contributory factor in the development of kidney cancer in the United States. This relationship is not as strong as that noted for the association between smoking and cancers of the lung, larynx, oral cavity, and esophagus. The term "contributory factor" by no means excludes the possibility of a causal role for smoking in cancers of this site.

Pancreatic Cancer

23. Cigarette smoking is a contributory factor in the development of pancreatic cancer in the United States. This relationship is not as strong as that noted for the association between smoking and cancers of the lung, larynx, oral cavity, and esophagus. The term "contributory factor" by no means excludes the possibility of a causal role for smoking in cancers of this site.

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Stomach Cancer

24. In epidemiological studies, an association between cigarette smoking and stomach cancer has been noted. The association is small in comparison with that noted for smoking and some other cancers.

Uterine Cervix Cancer

25. There are conflicting results in studies published to date on the existence of a relationship between smoking and cervical cancer; further research is necessary to define whether an association exists and, if so, whether that association is direct or indirect.

Mechanisms of Carcinogenesis

This overview presents evidence and observations on tobacco carcinogenesis primarily developed since 1978.

1. The biological activity of whole cigarette smoke and its tar and tar fractions can now be measured by improved inhalation assays in addition to tests for tumor-initiating, tumor-promoting, and cocarcinogenic activities on mouse skin.
2. Studies on smoke inhalation with the hamster now appear suitable for estimating the relative tumorigenic potential of whole smoke from commercial and experimental cigarettes. The identification of the smoke constituents that contribute to tumor induction in the respiratory tract is best achieved by fractionations of tar and by assays on mouse epidermis that determine the type and potency of the carcinogens. In combination with biochemical tests, mouse skin assays should also aid in evaluating the possible role of nicotine as a cocarcinogen.
3. The identification, formation, and metabolic activation of organ-specific carcinogens have been studied which help explain the increased risk to cigarette smokers of cancer of the esophagus, pancreas, kidney, and urinary bladder. In addition to certain aromatic amines, tobacco-specific N-nitrosamines appear to be an important group of organ specific carcinogens in tobacco and tobacco smoke. Little is known of the *in vivo* formation of organ-specific carcinogens from nicotine and other *Nicotiana* alkaloids. The modification of their enzymatic activation to ultimate carcinogenic forms needs to be explored by chemopreventive approaches.
4. Transplacental carcinogenesis as it may relate to effects of cigarette smoking should be investigated more fully. It has been known for some time that inhalation of tobacco smoke activates enzymes in the placenta and fetus and the consequences of such changes need to be studied.

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5. The continuing modification of U.S. cigarettes has led to changes in the quantitative and perhaps also the qualitative composition of the smoke. This ongoing development requires continued monitoring of the toxic and carcinogenic potential of the smoke of new cigarettes.
6. The changes in cigarette composition lead generally to reduced emission of major toxic mainstream smoke constituents as measured in analytical laboratories under machine-smoking conditions. Many smokers intensify puff volume and degree of inhalation when smoking a lower-yield cigarette. Therefore, it should be determined what effect different techniques of air dilution and filtration have in counteracting the increased smoke exposure that results from intensified smoking.
7. Snuff tobaccos are increasingly used as an alternative to cigarette smoking. More information is needed regarding the carcinogenic activity of snuff tobaccos and the presence of tumorigenic agents in these products.

Involuntary Smoking and Lung Cancer

1. Mainstream and sidestream cigarette smoke contain similar chemical constituents. (Mainstream smoke is smoke that the smoker inhales directly during puffing. Sidestream smoke is smoke emitted from a smoldering cigarette into the ambient air.) These constituents include known carcinogens, some of which are present in higher concentrations in sidestream smoke than they are in mainstream smoke. Passive or involuntary smoking differs from voluntary cigarette smoking with respect to the concentration of smoke components inhaled, the duration and frequency of smoke exposure, and the pattern of inhalation.
2. In two epidemiologic studies, an increased risk of lung cancer in nonsmoking wives of smoking husbands was found. In these studies, the nonsmoking wife's risk of lung cancer increased in relation to the extent of the husband's smoking. In a third study, the risk of lung cancer among nonsmoking wives of smoking husbands was also increased, but the difference was not statistically significant.
3. Although the currently available evidence is not sufficient to conclude that passive or involuntary smoking causes lung cancer in nonsmokers, the evidence does raise concern about a possible serious public health problem.

Cessation of Smoking

1. Ninety-five percent of those who have quit smoking have done so without the aid of an organized smoking cessation program, and most current smokers indicate a preference for quitting

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with a procedure they may use on their own, and a disinclination to enter an organized, comprehensive program.

2. Research evaluations of self-help aids have reported success rates up to 50 percent cessation at extended followups (6 to 15 months). Most estimates, however, fall below this, around 5 to 20 percent.
3. Brief and simple advice to quit smoking delivered by a physician has substantial potential for producing cessation in a cost-effective manner.
4. Televised smoking cessation clinics result in variable rates of abstinence at followup. The use of television and other mass media are a cost-effective intervention because of their large potential audiences.
5. Retrospective studies revealed greater use of self-reward and active problem-solving strategies among those who quit or reduced smoking on their own than among those who were unsuccessful in quitting or reducing smoking.
6. Until recently, the long-term outcome of intensive smoking cessation clinics has remained at 25 to 30 percent abstinence. New emphasis on techniques to improve the maintenance phase of cessation promises to improve these rates, with several reports of greater than 50 percent abstinence at followups of 6 months or longer.
7. To improve maintenance of nonsmoking after intensive treatment programs have ended, reinforcement should be built into the natural environment. Smoking cessation programs in the workplace may offer an opportunity for this.
8. Comprehensive self-management packages that have been shown to boost maintenance rates include a wide variety of techniques.
9. Treatment outcome may be improved by focusing on the antecedents of relapse. These include feelings of frustration, anxiety, anger, and depression as well as social models and smoking-related cues and settings. Behavioral and cognitive skills for dealing with such antecedents should be developed.
10. Social support interventions are promising. Reliable findings link social cues, smoking friends, and smoking spouses to relapse, whereas the presence of group support, nonsmoking spouses, and professional contact decreases recidivism.
11. Spontaneous smoking cessation among regular users (approximately once a week or more often) is estimated to be on the order of 25 percent during adolescence.
12. Probability of quitting was greater for those adolescent smokers first interviewed in 1974 who had at least started to attend college by 1979 than for those smokers who did not attend college (42.0 percent vs. 24.6 percent).

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13. Probability of quitting decreases linearly with duration of the smoking practice, changing from 64.5 percent in the first year of smoking to 14.3 percent after 7 years.
14. Quitting "cold turkey" appears to be a more effective cessation strategy than cutting down without trying to stop entirely.
15. Success at quitting increased with the number of efforts made: about 73.4 percent of adolescents who kept trying eventually succeeded.
16. Smoking prevention programs are desirable alternatives to cessation programs aimed at youth. Successful programs have been based on social psychological theory and research, and are school based. Results have shown a 50 percent or more reduction in smoking onset.
17. The most successful programs were those emphasizing the social and immediate consequences of smoking rather than long-term health consequences. These programs have placed special emphasis on teaching skills in recognizing and resisting social pressures to smoke.

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The Health Consequences Of Smoking

CARDIOVASCULAR DISEASE

a report of the Surgeon General

1983

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Washington, D.C. 20402

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THE SECRETARY OF HEALTH AND HUMAN SERVICES
WASHINGTON, D.C. 20201

NOV 17 1983

TO THE READERS OF THIS VOLUME:

Provisions of the Public Health Cigarette Smoking Act of 1969 (P.L. 91-222) require the Secretary of Health and Human Services to submit an annual report to the Congress on the health consequences of smoking. Attached is the 1983 report, Health Consequences of Smoking: Cardiovascular Disease. This volume is an indepth analysis of the scientific evidence of the relationship between cigarette smoking and multiple cardiovascular diseases. This relationship is quantitatively the most serious of the health consequences of smoking, but is poorly recognized by the public.

This report represents the consolidated work of many widely-recognized experts known for their contribution to understanding cardiovascular disease. It is a scientific reference document to serve as a state-of-the-art source for medical and behavioral scientists and researchers.

Smoking-related cardiovascular disease is estimated to account for more deaths than any other smoking-related disease, including cancer. This report clearly establishes that cigarette smoking increases the risks for a number of cardiovascular diseases, particularly coronary heart disease, the largest single cause of deaths in the United States. In addition, smoking is related to an increased risk for stroke, atherosclerosis, and other circulatory diseases.

The report clearly demonstrates that cigarette smoking is a major risk factor for coronary heart disease in the United States. There are 55 million persons who smoke, a larger population than those who have hypertension or elevated cholesterol, the other major risk factors for this disease. Smokers' death rates from coronary heart disease are 70 percent greater than those of non-smokers. Simply by quitting smoking, these men and women dramatically reduce their risk of premature death from this disease.

The economic and social toll these smoking-related deaths extract from the Nation's health is immeasurable. The report's findings re-emphasize the importance of this Department's continued educational efforts to enable a fully-informed choice by individuals on whether to begin or to continue to smoke.

In my view, this volume is a solid scientific work and a contribution to the prevention efforts of this Department.

Margaret M. Heckler

Margaret M. Heckler
Secretary

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SECTION 1. INTRODUCTION, OVERVIEW, AND CONCLUSIONS

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Introduction

Organization and Development of the 1983 Report

The content of the Report is the work of numerous scientists and experts within the Department of Health and Human Services as well as from outside the organization. Individual manuscripts were written by experts nationally and internationally recognized for their scientific contributions to the understanding of cardiovascular diseases. These manuscripts were reviewed individually by other experts, within and outside the U.S. Public Health Service, and the entire Report was reviewed by a broad-based panel of distinguished cardiovascular scientists. The 1983 Report includes a Foreword by the Assistant Secretary for Health of the Department of Health and Human Services and a Preface by the Surgeon General of the U.S. Public Health Service. The body of the report consists of eight sections and two appendices, as follows:

- Section 1. Introduction, Overview, and Conclusions
- Section 2. Arteriosclerosis
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Historical Perspective

Early reports linking smoking with a greater risk of developing cardiovascular disease occurred around the turn of the century. An early series of studies, initiated in 1904 by Erb, found a much higher percentage of smokers than of nonsmokers with intermittent claudication; only 10 percent of his patients with claudication were nonusers of tobacco. As early as 1934, Howard made the observation that the increasing prevalence of coronary heart disease noted since the first World War might be a result of the greatly increased use of cigarettes.

By the turn of the century, numerous studies had demonstrated clinically and experimentally that cigarette smoking or cigarette smoke constituents, most notably nicotine, caused an elevation in blood pressure and heart rate during smoking.

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The first major prospective study results were made public in 1954 in the United States by Hammond and Horn and found a strong association between cigarette use among men and coronary heart disease (CHD). Overall, smokers were found to carry a 70 percent greater risk of dying from CHD than nonsmokers; heavy smokers had CHD mortality rates almost two and one-half times greater than nonsmokers. Hammond and Horn also noted a consistent dose–response relationship with the number of cigarettes consumed per day.

In the intervening 30 years, numerous additional epidemiological mortality studies were undertaken to examine this issue. These included studies in the United Kingdom, Canada, Sweden, Japan, and Switzerland in addition to the United States. In total, they represent more than 20 million person-years of observation. Findings from these studies have been remarkably uniform: smokers have much higher death rates from coronary heart disease than do nonsmokers, despite the fact that these studies were conducted in varying populations, were geographically diverse, and involved differing methodologies.

The first major U.S. Public Health Service review of the relationship between smoking and heart disease was conducted by the Surgeon General's Advisory Committee on Smoking and Health in 1964. Although the Committee noted that male smokers had higher death rates from coronary heart disease, it was unable to conclude that the association had causal significance. However, it was noted in the report that "the causative role of these factors [risk factors including cigarette smoking] in coronary disease, though not proven, is suspected strongly enough to be a major reason for taking countermeasures against them. It is also more prudent to assume that the established association between cigarette smoking and coronary disease has causative meaning than to suspend judgement until no uncertainty remains."

Since the release of the original Report of the Surgeon General in 1964, additional studies dealing with cigarette smoking and CHD have been summarized in the series of annual reports of the Surgeon General *The Health Consequences of Smoking*. By 1979, the magnitude of the epidemiological, pathological, clinical, and experimental evidence had grown to the point that the Surgeon General's Report concluded: "Smoking is causally related to coronary heart disease in the common sense of that idea and for the purposes of preventive medicine."

Overview

In 1980, diseases of the circulatory system were responsible for approximately one-half of the total U.S. mortality. *CHD was the*

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single most important cause of death, accounting for approximately 30 percent of all U.S. deaths.

Cigarette smoking is one of the three major independent CHD risk factors. The magnitude of the risk associated with cigarette smoking is similar to that associated with the other two major CHD risk factors, hypertension and hypercholesterolemia; however, because cigarette smoking is present in a larger percentage of the U.S. population than either hypertension or hypercholesterolemia, cigarette smoking ranks as the largest preventable cause of CHD in the United States. Cigarette smoking also acts synergistically with the other major risk factors to greatly increase the risk for CHD.

Arteriosclerosis is the predominant underlying cause of cardiovascular disease, and atherosclerosis is the form of arteriosclerosis that most frequently causes clinically significant disease, including CHD, atherothrombotic brain infarction, atherosclerotic aortic disease, and atherosclerotic peripheral vascular disease. Cigarette smoking contributes both to the development of atherosclerotic lesions and to the clinical manifestations of atherosclerotic vascular disease, including sudden death. Although the precise pathophysiologic basis of these clinical manifestations is not understood, it may be related to several deleterious cardiovascular effects of cigarette smoking, including production of an imbalance between myocardial oxygen supply and demand, a decrease in the threshold for ventricular fibrillation, and an increase in platelet aggregation. Nicotine and carbon monoxide are the tobacco smoke constituents most closely associated with these adverse effects; other cigarette smoke constituents such as hydrogen cyanide, oxides of nitrogen, and carbon disulfide are being studied for possible pathogenic cardiovascular effects.

Cigarette smoking is the most important risk factor for atherosclerotic peripheral vascular disease, which usually involves the lower extremities. Smoking cessation is probably the single most important intervention in the management of this disorder. The effect of cigarette smoking to aggravate and accelerate the development of atherosclerosis is more striking in the aorta than in any other vessels. Cigarette smoking is associated with an increased risk for cerebrovascular disease, especially in younger age groups, but this effect is less marked than for atherosclerotic disease at other sites. Women cigarette smokers experience an increased risk for subarachnoid hemorrhage; the use of both cigarettes and oral contraceptives greatly increases this risk.

Smoking cessation is associated with decreased mortality and morbidity from atherosclerotic vascular disease. Prospective epidemiologic studies have shown that former cigarette smokers reduce their CHD death risk from that of current smokers to that of nonsmokers over approximately a 15-year period after stopping

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smoking. The beneficial effects of quitting are not explained by differences in baseline characteristics between quitters and continuing smokers. CHD intervention trials have successfully demonstrated the feasibility of reducing cigarette consumption; these trials also documented a significant reduction in CHD mortality.

Conclusions of the 1983 Report

The purpose of this Report is to review in depth the many sources of scientific evidence relating cigarette smoking to individual cardiovascular disease entities. Listed below are the major findings of this review.

Arteriosclerosis

1. A preponderance of evidence both from prospective studies with autopsy followup and from autopsy studies with retrospective smoking data indicates that cigarette smoking has a significant positive association with atherosclerosis. This evidence suggests that cigarette smoking has the effect of aggravating and accelerating the development of atherosclerotic lesions in the artery wall and that its effect is not limited to those events related to the occlusive episode. The effects are most striking for aortic atherosclerosis; a significant positive relationship also exists between cigarette smoking and atherosclerotic lesions in the coronary arteries, at least for most high risk populations. Cigarette smoking could also be associated with other factors that precipitate thrombosis, hemorrhage, or vasoconstriction leading to occlusion and ischemia.
2. Some evidence exists that cigarette smoke alters total serum cholesterol concentrations and lipoprotein composition in ways that would be expected to increase the development of atherosclerosis. Recent studies of the effects of smoking on the hemostatic system indicate effects on platelet function.
3. Although the specific mechanisms by which tobacco smoke affects arteriosclerosis have not been clearly delineated, the effects of cigarette smoking on the atherosclerotic lesions that underlie cardiovascular disease seem well established.

Coronary Heart Disease

1. Cigarette smoking is a major cause of coronary heart disease in the United States for both men and women. Because of the number of persons in the population who smoke and the increased risk that cigarette smoking represents, it should be considered the most important of the known modifiable risk factors for CHD.

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2. Overall, cigarette smokers experience a 70 percent greater CHD death rate than do nonsmokers. Heavy smokers, those who consume two or more packs per day, have CHD death rates between two and three times greater than nonsmokers.
3. The risk of developing CHD increases with increasing exposure to cigarette smoke, as measured by the number of cigarettes smoked daily, the total number of years one has smoked, and the degree of inhalation, and with an early age of initiation.
4. Cigarette smokers have a twofold greater incidence of CHD than do nonsmokers, and heavy smokers have an almost fourfold greater incidence.
5. Cigarette smoking is a major independent risk factor for CHD, and it acts synergistically with other risk factors (most notably, elevated serum cholesterol and hypertension) to greatly increase the risk of CHD.
6. Women have lower rates for CHD than do men. In particular, CHD rates for women are lower prior to the menopause. A part of this difference is due to the lower prevalence of smoking in women, and for those women who do smoke, to the tendency to smoke fewer cigarettes per day and to inhale less deeply. Among those women who have smoking patterns comparable to male smoking patterns, the increments in CHD death rates are similar for the two sexes.
7. Women who use oral contraceptives and who smoke increase their risk of a myocardial infarction by an approximately tenfold factor, compared with women who neither use oral contraceptives nor smoke.
8. Cigarette smoking has been found to significantly elevate the risk of sudden death. Overall, smokers experience a two to four times greater risk of sudden death than nonsmokers. The risk appears to increase with increasing dosage as measured by the number of cigarettes smoked per day and diminishes with cessation of smoking.
9. The CHD mortality ratio for smokers compared with nonsmokers is greater for the younger age groups than for the older age groups. Although the smoker-to-nonsmoker mortality ratio narrows with increasing age, smokers continue to experience greater CHD death rates at all ages.
10. Cigarette smoking has been estimated to be responsible for up to 30 percent of all CHD deaths in the United States each year. During the period 1965 to 1980 there were over 3 million premature deaths from heart disease among Americans attributed to cigarette smoking. Unless smoking habits of the American population change, perhaps 10 percent of all persons now alive may die prematurely of heart disease attributable to

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their smoking behavior. The total number of such premature deaths may exceed 24 million.

11. Cessation of smoking results in a substantial reduction in CHD death rates compared with those of persons who continue to smoke. Mortality from CHD declines rapidly after cessation. Approximately 10 years following cessation the CHD death rate for those ex-smokers who consumed less than a pack of cigarettes daily is virtually identical to that of lifelong nonsmokers. For ex-smokers who had smoked more than one pack per day, the residual risk of CHD mortality is proportional to the total lifetime exposure to cigarette smoke.
12. Epidemiologic evidence concerning reduced tar and nicotine or filter cigarettes and their effect on CHD rates is conflicting. No scientific evidence is available concerning the impact on CHD death rates of cigarettes with very low levels of tar and nicotine.
13. Smokers who have used only pipes or cigars do not appear to experience substantially greater CHD risks than nonsmokers.

Cerebrovascular Disease

1. Data from numerous prospective mortality studies have shown an association between cigarette smoking and cerebrovascular disease. This risk is most evident in the younger age groups, and the effect diminishes with increasing age, with little or no effect noted after age 65. No consistent dose–response effect has been demonstrated.
2. Women cigarette smokers experience an increased risk for subarachnoid hemorrhage. However, the use of both cigarettes and oral contraceptives greatly increases the risk for subarachnoid hemorrhage among women.

Atherosclerotic Peripheral Vascular Disease and Aortic Aneurysm

1. Cigarette smoking is the most powerful risk factor predisposing to atherosclerotic peripheral arterial disease.
2. Smoking cessation plays an important role in the medical and surgical management of atherosclerotic peripheral vascular disease.
3. Death from rupture of an atherosclerotic abdominal aneurysm is more common in cigarette smokers than in nonsmokers.

Pharmacological and Toxicological Implications of Smoke Constituents on Cardiovascular Disease

1. Over 4,000 different compounds have been identified in tobacco smoke.

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2. Nicotine exerts an effect on ganglionic cells, producing transient excitation. The pharmacological effects are small, but are reinforced several times daily in habitual smokers. The exact mechanisms whereby nicotine might influence cardiovascular events are unknown, but a lowering of the ventricular fibrillation threshold is dose related to nicotine levels.
3. Carbon monoxide may act to precipitate cardiac symptomatology or ischemic episodes in individuals already compromised by coronary disease. In addition, carbon monoxide binds to hemoproteins, potentially inhibiting their functions.
4. Several studies have shown that smokers may alter their smoking behavior when they switch to low-yield cigarettes. This compensatory behavior may lead to the increased uptake of gas phase constituents including carbon monoxide, hydrogen cyanide, and nitrous oxides.
5. It is unlikely that a "safe cigarette" can be developed that will reduce cardiovascular risk.

Changes in Cigarette Smoking Behavior in Clinical and Community Trials

1. Smokers involved in intervention programs demonstrate higher smoking cessation rates than those in control groups.
2. In general, the success of smoking intervention programs is related to the amount of intervention provided.

The Effect of Cigarette Smoking Cessation on Coronary Heart Disease

1. In the four intervention trials involving mortality followup of individual men for 5 to 10 years, the intervention groups had a combined total of 10 percent fewer CHD deaths than did the comparable control groups. Differences for other causes of death or for total deaths were not significant.
2. In these trials, the amount of cigarette smoking has been reduced 10 to 50 percent more in the intervention group than in the control group, demonstrating that intervention can alter smoking behavior.
3. In the two trials involving morbidity followup, the intervention groups had 4 and 45 percent lower total CHD incidence than did the respective control groups.
4. The relative reductions in CHD mortality in each of the four intervention studies involving individual followup are reasonably consistent with the reduction in CHD risk factors, and for a combination of all four studies, the reduction is statistically significant.

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5. Numerous studies have shown that those who quit cigarette smoking experience a substantial decrease in CHD mortality and an improvement in life expectancy.
6. A number of prospective epidemiological studies indicate that former cigarette smokers substantially reduce their CHD and total death rates from that of current smokers.

Trends in Cardiovascular Diseases

The evidence supports the conclusion that changes in smoking habits have contributed to substantial improvement in mortality rates from the cardiovascular diseases in the United States.

Trends in U.S. Cigarette Use, 1965–1980

1. The proportion of current regular smokers declined steadily between 1965 and 1980. The decline was steeper among males (from 52.1 to 37.9 percent) than among females (from 34.2 to 29.8 percent).
2. The proportion of never smokers increased steadily from 1965 to 1980 among males (27.6 to 31.6 percent), except those 45 years old and older. Among females, only 20- to 34-year-olds showed an increase in proportion of never smokers.
3. The mean number of cigarettes smoked per day by current smokers increased slightly from 1970 to 1980 (from 20 to 21.7 cigarettes).
4. Males smoked a higher mean number of cigarettes throughout the 1970–1980 period, but the number for males and females increased about the same amount.
5. Heaviest daily consumption was in the middle-aged group (35–65 years). The greatest mean increase was observed among women aged 35 to 44.
6. The proportion of current smokers who smoked less than 20 cigarettes per day decreased between 1970 and 1980 (39.8 to 33.8 percent); the proportion smoking one pack exactly (20 cigarettes) remained constant (34.9 to 34.8 percent); the proportion smoking from 21 to 39 cigarettes increased slightly (13.7 to 14.5 percent); and the proportion smoking two or more packs per day increased from 11.4 to 16.8 percent.
7. The proportion of current smokers who attempted to quit three or more times decreased slightly from 1966 to 1980 (41.2 to 38.7 percent).
8. The proportion of former smokers having made three or more attempts to quit increased sharply (36 to 53.2 percent) from 1966 to 1975.
9. The proportion of current smokers who had attempted to quit during the past year increased from 1966 to 1980 (26.0 to 36.7 percent).

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10. Among current smokers, younger persons and females were more likely than older persons and males to have attempted to quit during the previous 12 months.
11. The proportion of former smokers who had attempted to quit during the previous 12 months decreased from 1966 to 1975 (13.8 to 9.8 percent).
12. Among former smokers, younger persons and females were more likely than older persons and males to have quit during the previous 12 months.

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The Health Consequences Of Smoking

CHRONIC OBSTRUCTIVE LUNG DISEASE

a report of the Surgeon General

1984

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THE SECRETARY OF HEALTH AND HUMAN SERVICES
WASHINGTON, D.C. 20201

The Honorable Thomas P. O'Neill, Jr.
Speaker of the House of Representatives
Washington, D.C. 20515

Dear Mr. Speaker:

It is a pleasure to transmit to the Congress the Surgeon General's Report on the Health Consequences of Smoking, as mandated by Section 8(a) of the Public Health Cigarette Smoking Act of 1969. This is the Public Health Services' 16th report on this topic and, like all of the earlier Reports, it identifies cigarette smoking as the chief preventable cause of death and disability in our society.

The enclosed report deals with the relationship between smoking and those disease conditions described as chronic obstructive lung disease, particularly chronic bronchitis and emphysema. These diseases significantly increase patient loads in hospitals and other health care facilities and escalate this Nation's health care costs, including expenditures under the Medicaid and Medicare programs.

This report indicates that chronic obstructive lung diseases can be reduced and, in the case of emphysema, almost eradicated, if individuals stop cigarette smoking. Moreover, stopping smoking also would prevent the enormous suffering and human loss now well-known to be associated with smoking.

This Department has a strong and ongoing commitment to its programmatic and research efforts in the field of disease prevention. In our view, it is essential to apprise individuals of the consequences of smoking. A central part of our efforts is to identify ways to help smokers quit smoking, and to encourage individuals, particularly the youth of this country, not to begin smoking.

Sincerely,

Margaret M. Heckler

Margaret M. Heckler
Secretary

Enclosure

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THE SECRETARY OF HEALTH AND HUMAN SERVICES,
WASHINGTON, D.C. 20201

The Honorable George Bush
President of the Senate
Washington, D.C. 20510

Dear Mr. President:

It is a pleasure to transmit to the Congress the enclosed General's Report on the Health Consequences of Smoking, as mandated by Section 8(a) of the Public Health Cigarette Smoking Act of 1969. This is the Public Health Service's [illegible] report this topic and, like all of the earlier Reports, [illegible] cigarette smoking as the chief preventable cause of death and disability in our society.

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This Department has a strong and ongoing commitment to its programmatic and research efforts in the field of disease prevention. In our view, it is essential to apprise individuals of the consequences of smoking. A central part of our efforts [illegible] identify ways to help smokers quit smoking, and to encourage individuals, particularly the youth of this country, [illegible] smoking.

Sincerely,

Margaret M. Heckler

Margaret M. Heckler
Secretary

Enclosure

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CHAPTER 1. INTRODUCTION, OVERVIEW, AND CONCLUSIONS

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- Community Studies of Smoking Cessation and Prevention

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Introduction

Organization and Development of the 1984 Report

Each year the Office on Smoking and Health (OSH), working in close collaboration with scientists, researchers, and others, compiles the annual Surgeon General's Report *The Health Consequences of Smoking* for submission to the U.S. Congress as part of the Department's responsibility to report new and current information on the topic as required under Public Law 91-222. This Report is the third to examine in detail specific disease entities related to smoking. The 1982 Report was a comprehensive assessment of the relationship between tobacco use and various cancers, and the 1983 Report examined this relationship for cardiovascular diseases. The 1984 volume represents a state-of-the-art comprehensive review of tobacco use and the development of chronic obstructive lung diseases.

The scientific content of this Report is the work of experts in the field of chronic obstructive lung disease research both within the Department of Health and Human Services and from outside the Federal Government. Individual manuscripts were written by experts who are nationally and internationally recognized for their scientific understanding of the etiology of chronic obstructive lung diseases, particularly the relationship with cigarette use.

Manuscripts received from authors were extensively reviewed by numerous outside experts familiar with these specific areas. The entire Report was then submitted to a broad-based panel of 11 distinguished lung disease experts and to experts within the U.S. Public Health Service for their review and comments.

The 1984 Report includes a Foreword by the Assistant Secretary for Health of the Department of Health and Human Services and a Preface by the Surgeon General of the U.S. Public Health Service. The body of the Report consists of 10 chapters, as follows:

- Chapter 1. Introduction, Overview, and Conclusions
- Chapter 2. Effect of Cigarette Smoke Exposure on Measures of Chronic Obstructive Lung Disease Morbidity
- Chapter 3. Mortality From Chronic Obstructive Lung Disease Due to Cigarette Smoking
- Chapter 4. Pathology of Lung Disease Related to Smoking
- Chapter 5. Mechanisms by Which Cigarette Smoke Alters the Structure and Function of the Lung
- Chapter 6. Low Yield Cigarettes and Their Role in Chronic Obstructive Lung Disease
- Chapter 7. Passive Smoking
- Chapter 8. Deposition and Toxicity of Tobacco Smoke in the Lung

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- Chapter 9. Role of the Physician in Smoking Cessation
- Chapter 10. Community Studies of Smoking Cessation and Prevention

Historical Perspective

The relationship between cigarette smoking and chronic obstructive lung disease (COLD) was among the first recognized and is now the best understood of the diseases caused by smoking. Sigmund reported as early as 1870 that heavy smokers suffered "affections" of the nose, mouth, and throat more frequently and in a more virulent fashion. In 1897, Mendelssohn reported the incidence of "affections" of the respiratory tract to be 60 percent greater in smokers than in nonsmokers, as well as somewhat greater in those who inhaled compared with smokers who did not inhale.

Overview

Scientists from a variety of disciplines have investigated the role of cigarette smoking in the development of COLD; today we can trace the progressive decline in lung function in smokers with increasing smoke exposure, describe the concurrent pathologic changes, demonstrate that both COLD prevalence and COLD death are limited largely to smokers, and describe in detail a plausible mechanism by which cigarette smoking can lead to the development of emphysema. Some gaps in the understanding of the details of this process may still exist, but the experimental and epidemiologic evidence leaves no room for reasonable doubt on the fundamental issue: cigarette smoking is the major cause of COLD in the United States.

The earliest recognized response to cigarette smoke is an increase in airway resistance that occurs with the inhalation of smoke by the smoker. This increase in resistance is a response to the irritants in the smoke, as is coughing, which is more frequent in smokers than in nonsmokers, even among adolescents. By the time smokers become young adults, a substantial proportion of them will have developed pathologic changes in their small airways. These abnormalities are demonstrable using a variety of physiologic tests, and are a result of pathologic changes or inflammation in the airways less than 2 mm in diameter. Part of this small airways response, but perhaps a later manifestation of it, is the development of smooth muscle hypertrophy, goblet cell hyperplasia, and mild peribronchiolar fibrosis. The prevalence of abnormalities on tests of small airways function increases as these young smokers grow older, and is greater in heavy smokers than in light smokers. While it is clear that changes in the small airways represent an early response to cigarette smoking and that they are a significant finding in the pathophysiology of COLD, it is not clear that abnormal function of the small airways, per se, is

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useful as a marker for identifying who will progress to develop symptomatic COLD. It may identify a large group of smokers who manifest an irritant response to smoke in the small airways, of whom only a subset actually develop symptomatic airflow obstruction.

Measurable differences in tests of expiratory airflow exist between smokers and nonsmokers after age 25. Smokers as a group have a more rapid decline in FEV_1 with age than that observed in nonsmokers, and the decline is even greater among heavy smokers. However, this increased rate of decline in lung function is not distributed evenly, even among smokers with similar smoking histories. Some smokers have a far more rapid decline than the average smoker, and clearly those individuals who have developed symptomatic chronic airflow obstruction have had a larger total decline in lung function than the average smoker. This has led to the suggestion that individuals with a particularly rapid decline in FEV_1 early in life may represent a group especially susceptible to the later development of symptomatic COLD. The nature of this susceptibility remains unclear, but differences in depth or pattern of inhalation, variations in the cellular and biochemical response of the lung to smoke, differences in immune or repair mechanisms, and childhood infections or exposure to environmental tobacco smoke as a child have been suggested as potential factors.

The accumulation of lung damage, marked by the excess decline in FEV_1 and other measures of expiratory airflow, can lead to shortness of breath and other symptoms that characterize clinically significant COLD. These symptoms can result in disability due to ventilatory limitation and may vary from patient to patient in severity and duration. Many patients with clinically disabling COLD die with the disease rather than because of it. Death from COLD usually results only after extensive lung damage and commonly occurs because of failure of the severely damaged lungs to maintain adequate gas exchange.

The cessation of cigarette smoking has a substantial salutary impact on the incidence and progression of COLD. Cigarette smokers who quit prior to developing abnormal lung function are unlikely to go on to develop ventilatory limitation; when the abnormalities are demonstrable only on tests of small airways function, cessation often results in a reversal of these changes and a return to normal function. The presence of significant fixed reduction in measures of expiratory airflow usually reflects the presence of substantial lung damage. Cessation of smoking at this stage of COLD results in a slowing in the rate of decline in lung function with age, in comparison with that in continuing smokers. After a period of cessation, this rate of decline in function may approximate the rate found in nonsmokers, but there is little evidence to suggest that

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those who quit are able to regain their prior excess functional loss. Therefore, those who quit continue to have reduced lung function when compared with those who have never smoked, but their lung function begins to decline less rapidly with age when compared to the lung function of those who continue to smoke.

The importance of cigarette smoking as a causative factor in COLD is emphasized by cross-sectional studies of populations in the United States where often the only major predictor for developing or dying of COLD is smoking behavior. In the absence of cigarette smoking, clinically significant COLD is rare.

As the smoker enters the sixth decade of life, pathologically definable pulmonary emphysema begins to become evident. In older age groups, mild to moderate emphysema is present in most smokers and is rare in nonsmokers. Once again, however, only a small percentage of smokers develop severe emphysema; this minority includes a disproportionate number of heavy smokers.

A mechanism for smoking-induced emphysematous lung injury has been proposed and continues to evolve as our understanding of cellular and biochemical responses of the lung increases. Emphysema can be produced by the presence of excessive amounts of elastase (an enzyme capable of degrading the structural elements of lung tissue) or by the absence of α_1-antiprotease (a protein that inhibits the action of elastase). As part of the inflammatory response to cigarette smoke, an increased number of inflammatory cells are present in the lungs of smokers; these cells may result in an increased amount of elastase being present in the lung. In addition, cigarette smoke can oxidize the α_1-antiprotease in the lung, further contributing to the imbalance between levels of elastase and levels of α_1-antiprotease. The net result can be excess elastase activity, leading to degradation of elastin in the lung, destruction of alveolar walls, and development of emphysema.

The text of this Report discusses in detail the relationship of cigarette smoking to COLD morbidity and mortality, the pathology of smoking-induced COLD, some of the mechanisms by which smoking results in COLD, the impact on the lung of low tar and nicotine cigarettes and of involuntary smoke exposure, the deposition and toxicology of tobacco smoke, and the role of the physician and of community intervention programs in smoking cessation.

The overall conclusion of this Report is clear: **Cigarette smoking is the major cause of chronic obstructive lung disease in the United States for both men and women. The contribution of cigarette smoking to chronic obstructive lung disease morbidity and mortality far outweighs all other factors.**

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demonstrated a more extensive decline in lung function than the average smoker. The data are not yet available to determine whether a rapid decline in lung function early in life defines the subgroup of smokers who are susceptible to developing COLD.

10. Clinically significant degrees of emphysema occur almost exclusively in cigarette smokers or individuals with genetic homozygous α_1-antiprotease deficiency. The severity of emphysema among smokers increases with the number of cigarettes smoked per day and the duration of the smoking habit.

COLD Mortality

1. Data from both prospective and retrospective studies consistently demonstrate a uniform increase in mortality from COLD for cigarette smokers compared with nonsmokers. Cigarette smoking is the major cause of COLD mortality for both men and women in the United States.
2. The death rate from COLD is greater for men than for women, most likely reflecting the differences in lifetime smoking patterns, such as a smaller percentage of women smoking in past decades, and their smoking fewer cigarettes, inhaling less deeply, and beginning to smoke later in life.
3. Differences in lifetime smoking behavior are less marked for younger age cohorts of smokers. The ratio of male to female mortality from COLD is decreasing because of a more rapid rise in mortality from COLD among women.
4. The dose of tobacco exposure as measured by number of cigarettes or duration of habit strongly affects the risk for death from COLD in both men and women. Similarly, people who inhale deeply experience an even higher risk for mortality from COLD than those who do not inhale.
5. Cessation of smoking leads eventually to a decreased risk of mortality from COLD, compared with that of continuing smokers. The residual excess risk of death for the ex-smoker is directly proportional to the overall lifetime exposure to cigarette smoke and to the total number of years since one quit smoking. However, the risk of COLD mortality among former smokers does not decline to equal that of the never smoker even after 20 years of cessation.
6. Several prospective epidemiologic studies examined the relationship between pipe and cigar smoking and mortality from COLD. Pipe smokers and cigar smokers also experience higher mortality from COLD compared with nonsmokers; however, the risk is less than that for cigarette smokers.
7. There are substantial worldwide differences in mortality from COLD. Some of these differences are due to variations in

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Conclusions of the 1984 Report

COLD Morbidity

1. Cigarette smoking is the major cause of COLD morbidity in the United States; 80 to 90 percent of COLD in the United States is attributable to cigarette smoking.
2. In population-based studies in the United States, cigarette smoking behavior is often the only significant predictor for the development of COLD. Other factors improve the predictive equation only slightly, even in those populations where they have been found to exert a statistically significant effect.
3. In spite of over 30 years of intensive investigation, only cigarette smoking and α_1-antiprotease deficiency (a rare genetic defect) are established causes of clinically significant COLD in the absence of other agents.
4. Within a few years after beginning to smoke, smokers experience a higher prevalence of abnormal function in the small airways than nonsmokers. The prevalence of abnormal small airways function increases with age and the duration of the smoking habit, and is greater in heavy smokers than in light smokers. These abnormalities in function reflect inflammatory changes in the small airways and often reverse with the cessation of smoking.
5. Both male and female smokers develop abnormalities in the small airways, but the data are not sufficient to define possible sex-related differences in this response. It seems likely, however, that the contribution of sex differences is small when age and smoking exposure are taken into account.
6. There is, as yet, inadequate information to allow a firm conclusion to be drawn about the predictive value of the tests of small airways function in identifying the susceptible smoker who will progress to clinical airflow obstruction.
7. Smokers of both sexes have a higher prevalence of cough and phlegm production than nonsmokers. This prevalence increases with an increasing number of cigarettes smoked per day and decreases with the cessation of smoking.
8. Differences between smokers and nonsmokers in measures of expiratory airflow are demonstrable by young adulthood and increase with number of cigarettes smoked per day.
9. The rate of decline in measures of expiratory airflow with increasing age is steeper for smokers than for nonsmokers; it is also steeper for heavy smokers than for light smokers. After the cessation of smoking, the rate of decline of lung function with increasing age appears to slow to approximately that seen in nonsmokers of the same age. Only a minority of smokers will develop clinically significant COLD, and this group will have

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Pathology of Cigarette-Induced Disease

1. Smoking induces changes in multiple areas of the lung, and the effects in the different areas may be independent of each other. In the bronchi (the large airways), smoking results in a modest increase in size of the tracheobronchial glands, associated with an increase in secretion of mucus, and in an increased number of goblet cells.
2. In the small airways (conducting airways 2 or 3 mm or less in diameter consisting of the smallest bronchi and bronchioles) a number of lesions are apparent. The initial response to smoking is probably inflammation, with associated ulceration and squamous metaplasia. Fibrosis, increased muscle mass, narrowing of the airways, and an increase in the number of goblet cells follow.
3. Inflammation appears to be the major determinant of small airways dysfunction and may be reversible after cessation of smoking.
4. The most obvious difference between smokers and nonsmokers is respiratory bronchiolitis. This lesion may be an important cause of abnormalities in tests of small airways function, and may be involved in the pathogenesis of centrilobular emphysema. The severity of emphysema is clearly associated with smoking, and severe emphysema is confined largely to smokers.

Mechanisms of COLD

1. Increased numbers of inflammatory cells are found in the lungs of cigarette smokers. These cells include macrophages and, probably, neutrophils, both of which can release elastase in the lung.
2. Human neutrophil elastase produces emphysema when instilled into animal lungs.
3. Alpha$_1$-antiprotease inhibits the action of elastase, and a very small number of people with a homozygous deficiency of α_1-antiprotease are at increased risk of developing emphysema. The α_1-antiprotease activity has been shown to be reduced in the bronchoalveolar fluids obtained from cigarette smokers and from rats exposed to cigarette smoke.
4. The protease–antiprotease hypothesis suggests that emphysema results when there is excess elastase activity as the result of increased concentrations of inflammatory cells in the lung

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and of decreased levels of α_1-antiprotease secondary to oxidation by cigarette smoke.

5. Cigarette smokers have been shown to have a more rapid fall in antibody levels following immunization for influenza than nonsmokers. Whole cigarette smoke has been shown to depress the number of antibody-forming cells in the spleens of experimental animals.
6. Cigarette smoke produces structural and functional abnormalities in the airway mucociliary system.
7. Short-term exposure to cigarette smoke causes ciliostasis in vitro, but has inconsistent effects on mucociliary function in man. Long-term exposure to cigarette smoke consistently causes an impairment of mucociliary clearance. This impairment is associated with epithelial lesions, mucus hypersecretion, and ciliary dysfunction.
8. Chronic bronchitis in smokers and ex-smokers is characterized by an impairment of mucociliary clearance.
9. Both the particulate phase and the gas phase of cigarette smoke are ciliotoxic.

Low Tar and Nicotine Cigarettes

1. The recommendation for those who cannot quit to switch to smoking cigarette brands with low tar and nicotine yields, as determined by a smoking-machine, is based on the assumption that this switch will result in a reduction in the exposure of the lung to these toxic substances. The design of the cigarette has markedly changed in recent years, and this may have resulted in machine-measured tar and nicotine yields that do not reflect the real dose to the smoker.
2. Smoking-machines that take into account compensatory changes in smoking behavior are needed. The assays could provide both an average and a range of tar and nicotine yields produced by different individual patterns of smoking.
3. Although a reduction in cigarette tar content appears to reduce the risk of cough and mucus hypersecretion, the risk of shortness of breath and airflow obstruction may not be reduced. Evidence is unavailable on the relative risks of developing COLD consequent to smoking cigarettes with the very low tar and nicotine yields of current and recently marketed brands.
4. Smokers who switch from higher to lower yield cigarettes show compensatory changes in smoking behavior: the number of puffs per cigarette is variably increased and puff volume is almost universally increased, although the number of cigarettes smoked per day and inhalation volume are generally

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unchanged. Full compensation of dose for cigarettes with lower yields is generally not achieved.

5. Nicotine has long been regarded as the primary reinforcer of cigarette smoking, but tar content may also be important in determining smoking behavior.
6. Depth and duration of inhalation are among the most important factors in determining the relative concentration of smoke constituents that reach the lung. Considerable interindividual variation exists between smokers with respect to the volume and duration of inhalation. This variation is likely to be an important factor in determining the varying susceptibility of smokers to the development of lung disease.
7. Production of low tar and nicotine cigarettes has progressed beyond simple reduction in tobacco content. Additives such as artificial tobacco substitutes and flavoring extracts have been used. The identity, chemical composition, and adverse biological potential of these additives are unknown at present.

Passive Smoking

1. Cigarette smoke can make a significant, measurable contribution to the level of indoor air pollution at levels of smoking and ventilation that are common in the indoor environment.
2. Nonsmokers who report exposure to environmental tobacco smoke have higher levels of urinary cotinine, a metabolite of nicotine, than those who do not report such exposure.
3. Cigarette smoke in the air can produce an increase in both subjective and objective measures of eye irritation. Further, some studies suggest that high levels of involuntary smoke exposure might produce small changes in pulmonary function in normal subjects.
4. The children of smoking parents have an increased prevalence of reported respiratory symptoms, and have an increased frequency of bronchitis and pneumonia early in life.
5. The children of smoking parents appear to have measurable but small differences in tests of pulmonary function when compared with children of nonsmoking parents. The significance of this finding to the future development of lung disease is unknown.
6. Two studies have reported differences in measures of lung function in older populations between subjects chronically exposed to involuntary smoking and those who were not. This difference was not found in a younger and possibly less exposed population.
7. The limited existing data yield conflicting results concerning the relationship between passive smoke exposure and pulmonary function changes in patients with asthma.

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Deposition and Toxicity of Tobacco Smoke in the Lung

1. The mass median aerodynamic diameter of the particles in cigarette smoke has been measured to average approximately 0.46 μm, and particulate concentrations have been shown to range from 0.3×10^9 to 3.3×10^9 per milliliter.
2. The particulate concentration of the smoke increases as the cigarette is more completely smoked.
3. Particles in the size range of cigarette smoke will deposit both in the airways and in alveoli; models predict that 30 to 40 percent of the particles within the size range present in cigarette smoke will deposit in alveolar regions and 5 to 10 percent will deposit in the tracheobronchial region.
4. Acute exposure to cigarette smoke results in an increase in airway resistance in both animals and humans.
5. Exposure to cigarette smoke results in an increase in pulmonary epithelial permeability in both humans and animals.
6. Cigarette smoke has been shown to impair elastin synthesis in vitro and elastin repair in vivo in experimental animals (elastin is a vital structural element of pulmonary tissue).

Role of the Physician in Smoking Cessation

1. At least 70 percent of North Americans see a physician once a year. Thus, an estimated 38 million of the 54 million adults in the United States who smoke cigarettes could be reached annually with a smoking cessation message by their physician.
2. Current smoking prevalence among physicians in the United States is estimated at 10 percent.
3. While the majority of persons who smoke feel that physician advice to quit or cut down would be influential, there is a disparity between physicians' and patients' estimates of cessation counseling, with physician advice being reported by only approximately 25 percent of current smokers.
4. Studies of routine (minimal) advice to quit smoking delivered by general practitioners have shown sustained quit rates of approximately 5 percent. Followup discussions enhance the effects of physician advice.
5. A median of 20 percent of pregnant women who smoke quit spontaneously during pregnancy. That proportion can be doubled by an intervention consisting of health education, behavioral strategies, and multiple contacts.
6. Large controlled trials of cardiovascular risk reduction have demonstrated that counseling on individual specific risk factors, including smoking cessation techniques, can be effective.
7. Studies of pulmonary and cardiac patients indicate that severity of illness is positively related to increased compliance.

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8. Nicotine chewing gum has been developed as a pharmacological aid to smoking cessation, primarily to alleviate withdrawal symptoms. Cessation studies conducted in offices of physicians who prescribe the gum have produced mixed results, however, with outcome depending on motivation and intensity of adjunctive support or followup.
9. Physician-assisted intervention quit rates vary according to the type of intervention, provider performance, and patient group. In general, quit rates in recent research appear to be lower than in older studies.

Community Studies of Smoking Cessation and Prevention

1. Community studies of smoking cessation and prevention are becoming an established paradigm for public health action research. Such studies emphasize large-scale delivery systems, such as the mass media, and include community organization programs seeking to stimulate interpersonal communication in ways that are feasible on a large-scale basis.
2. Although there are methodological limitations to nearly all communitywide studies, the results yield fairly consistent positive results, indicating that large-scale programs to reduce smoking can be effective in whole populations. Person-to-person communication appears to be a necessary part of a successful community program to reduce smoking.
3. Further research is needed, with both improved methodology and more emphasis on low socioeconomic status groups that have not yet shown population trends toward reduced smoking.
4. Several promising directions for research are clear, but the most important future trends will be toward the establishment of smoking reduction programs within existing health services, the combination of chronic disease prevention with mental health promotion via mass media and community intervention, and the development of social policy to establish integrated strategies for smoking cessation and prevention.

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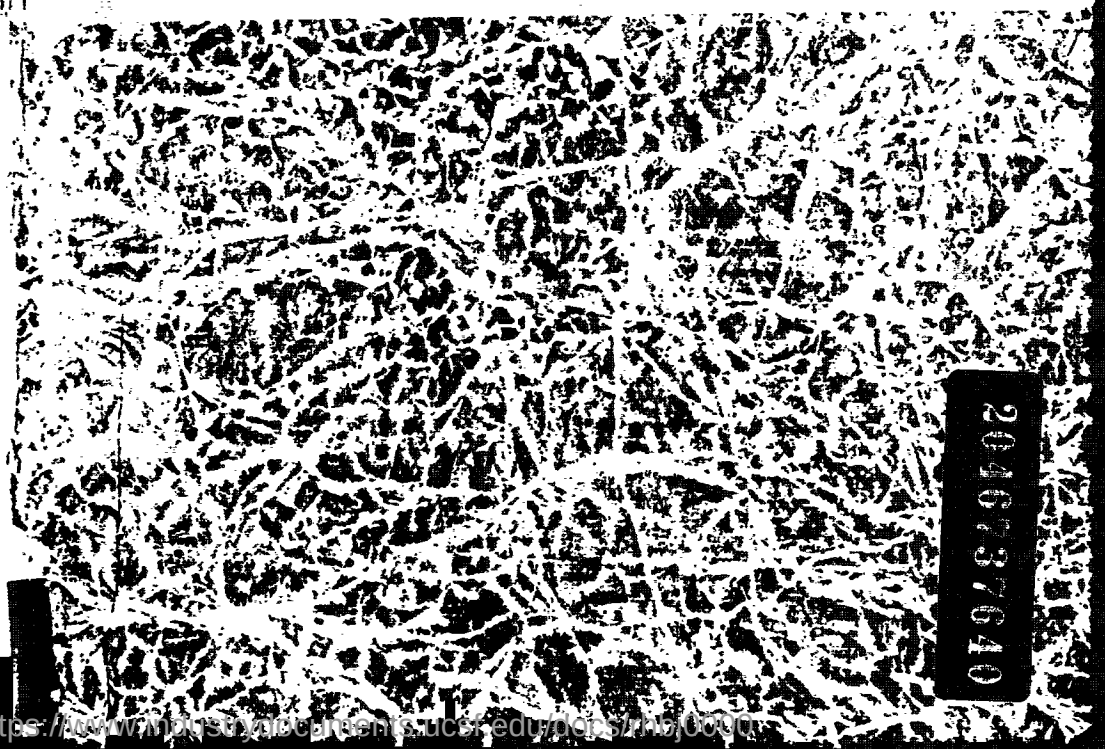
SMOKING *and* HEALTH

REPORT OF THE ADVISORY COMMITTEE
TO THE SURGEON GENERAL
OF THE PUBLIC HEALTH SERVICE

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service



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Chapter 4

Summaries and Conclusions

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Chapter 4

This chapter is presented in two sections. Section A contains background information, the gist of the Committee's findings and conclusions on tobacco and health, and an assessment of the nature and magnitude of the health hazard. Section B presents all formal conclusions adopted by the Committee and selected comments abridged from the detailed Summaries that appear in each chapter of Part II of the Report. The full scope and depth of the Committee's inquiry may be comprehended only by study of the complete Report.

A. BACKGROUND AND HIGHLIGHTS

In previous studies, the use of tobacco, especially cigarette smoking, has been causally linked to several diseases. Such use has been associated with increased deaths from lung cancer and other diseases, notably coronary artery disease, chronic bronchitis, and emphysema. These widely reported findings, which have been the cause of much public concern over the past decade, have been accepted in many countries by official health agencies, medical associations, and voluntary health organizations.

The potential hazard is great because these diseases are major causes of death and disability. In 1962, over 500,000 people in the United States died of arteriosclerotic heart disease (principally coronary artery disease), 41,000 died of lung cancer, and 15,000 died of bronchitis and emphysema.

The numbers of deaths in some important disease categories that have been reported to have a relationship with tobacco use are shown in Table 1. This table presents one aspect of the size of the potential hazard; the degree of association with the use of tobacco will be discussed later.

Another cause for concern is that deaths from some of these diseases have been increasing with great rapidity over the past few decades.

Lung cancer deaths, less than 3,000 in 1930, increased to 18,000 in 1950. In the short period since 1955, deaths from lung cancer rose from less than 27,000 to the 1962 total of 41,000. This extraordinary rise has not been recorded for cancer of any other site. While part of the rising trend for lung cancer is attributable to improvements in diagnosis and the changing age-composition and size of the population, the evidence leaves little doubt that a true increase in lung cancer has taken place.

Deaths from arteriosclerotic, coronary, and degenerative heart disease rose from 273,000 in 1940, to 396,000 in 1950, and to 578,000 in 1962.

Reported deaths from chronic bronchitis and emphysema rose from 2,300 in 1945 to 15,000 in 1962.

The changing patterns and extent of tobacco use are a pertinent aspect of the tobacco-health problem.

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TABLE 1.—*Deaths from selected disease categories, United States, 1962*

Cause of death*	Total	Males	Females
Degenerative and arteriosclerotic heart disease, including coronary disease (420, 422)	[illegible]	348,664	[illegible]
Hypertensive heart disease (440-3)	[illegible]	[illegible]	[illegible]
Cancer of lung (162-3)	[illegible]	[illegible]	[illegible]
Cirrhosis of liver (581)	[illegible]	[illegible]	[illegible]
Bronchitis and emphysema (502, 527.1)	[illegible]	12,337	[illegible]
Stomach and duodenal ulcers (540-1)	[illegible]	[illegible]	[illegible]
Cancer of bladder (181)	[illegible]	[illegible]	[illegible]
Cancer of oral cavity (140-8)	[illegible]	[illegible]	[illegible]
Cancer of esophagus (150)	[illegible]	[illegible]	[illegible]
Cancer of larynx (161)	[illegible]	[illegible]	[illegible]
All above causes	[illegible]	[illegible]	[illegible]
All other causes	1,004,027	531,477	472,550
All causes	1,756,720	994,789	[illegible]

*International Statistical Classification numbers in parentheses.

Nearly 70 million people in the United States consume tobacco regularly. Cigarette consumption in the United States has increased markedly since the turn of the Century, when per capita consumption was less than 50 cigarettes a year. Since 1910, when cigarette consumption per person (15 years and older) was 138, it rose to 1,365 in 1930, to 1,828 in 1940, to 3,322 in 1950, and to a peak of 3,986 in 1961. The 1955 Current Population Survey showed that 68 percent of the male population and 32.4 percent of the female population 18 years of age and over were regular smokers of cigarettes.

In contrast with this sharp increase in cigarette smoking, per capita use of tobacco in other forms has gone down. Per capita consumption of cigars declined from 117 in 1920 to 55 in 1962. Consumption of pipe tobacco, which reached a peak of 2½ lbs. per person in 1910, fell to a little more than half a pound per person in 1962. Use of chewing tobacco has declined from about four pounds per person in 1900 to half a pound in 1962.

The background for the Committee's study thus included much general information and findings from previous investigations which associated the increase in cigarette smoking with increased deaths in a number of major disease categories. It was in this setting that the Committee began its work to assess the nature and magnitude of the health hazard attributable to smoking.

KINDS OF EVIDENCE

In order to judge whether smoking and other tobacco uses are injurious to health or related to specific diseases, the Committee evaluated three main kinds of scientific evidence:

1. *Animal experiments.*—In numerous studies, animals have been exposed to tobacco smoke and tars, and to the various chemical compounds they contain. Seven of these compounds (polycyclic aromatic compounds) have been established as cancer-producing (carginogenic). Other substances in tobacco and smoke, though not carcinogenic themselves, promote cancer production or lower the threshold to a known carcinogen. Several toxic or irritant gases contained in tobacco smoke produce experimentally the kinds of non-cancerous damage seen in the tissues and cells of heavy smokers. This includes

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suppression of ciliary action that normally cleanses the trachea and bronchi, damage to the lung air sacs, and to mucous glands and goblet cells which produce mucus.

2. *Clinical and autopsy studies.*—Observations of thousands of patients and autopsy studies of smokers and non-smokers show that many kinds of damage to body functions and to organs, cells, and tissues occur more frequently and severely in smokers. Three kinds of cellular changes—loss of ciliated cells, thickening (more than two layers of basal cells), and presence of atypical cells—are much more common in the lining layer (epithelium) of the trachea and bronchi of cigarette smokers than of non-smokers. Some of the advanced lesions seen in the bronchi of cigarette smokers are probably premalignant. Cellular changes regularly found at autopsy in patients with chronic bronchitis are more often present in the bronchi of smokers than non-smokers. Pathological changes in the air sacs and other functional tissue of the lung (parenchyma) have a remarkably close association with past history of cigarette smoking.

3. *Population studies.*—Another kind of evidence regarding an association between smoking and disease comes from epidemiological studies.

In retrospective studies, the smoking histories of persons with a specified disease (for example, lung cancer) are compared with those of appropriate control groups without the disease. For lung cancer alone, 29 such retrospective studies have been made in recent years. Despite many variations in design and method, all but one (which dealt with females) showed that proportionately more cigarette smokers are found among the lung cancer patients than in the control populations without lung cancer.

Extensive retrospective studies of the prevalence of specific symptoms and signs—chronic cough, sputum production, breathlessness, chest illness, and decreased lung function—consistently show that these occur more often in cigarette smokers than in non-smokers. Some of these signs and symptoms are the clinical expressions of chronic bronchitis, and some are associated more with emphysema; in general, they increase with amount of smoking and decrease after cessation of smoking.

Another type of epidemiological evidence on the relation of smoking and mortality comes from seven prospective studies which have been conducted since 1951. In these studies, large numbers of men answered questions about their smoking or non-smoking habits. Death certificates have been obtained for those who died since entering the studies, permitting total death rates and death rates by cause to be computed for smokers of various types as well as for non-smokers. The prospective studies thus add several important dimensions to information on the smoking-health problem. Their data permit direct comparisons of the death rates of smokers and non-smokers, both overall and for individual causes of death, and indicate the strength of the association between smoking and specific diseases.

Each of these three lines of evidence was evaluated and then considered together in drawing conclusions. The Committee was aware that the mere establishment of a statistical association between the use of tobacco and a disease is not enough. The causal significance of the use of tobacco in relation to the disease is the crucial question. For such judgments all three

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lines of evidence are essential, as discussed in more detail on pages 26–27 of this Chapter, and in Chapter 3.

The experimental, clinical, and pathological evidence, as well as data from population studies, is highlighted in Section B of this Chapter, which in turn refers the reader to specific places in Part II of the Report where this evidence is presented in detail.

In the paragraphs which follow, the Committee has chosen to summarize the results of the seven prospective population studies which, as noted above, constitute only one type of evidence. They illustrate the nature and potential magnitude of the smoking-health problem, and bring out a number of factors which are involved.

Evidence From the Combined Results of Prospective Studies

The Committee examined the seven prospective studies separately as well as their combined results. Considerable weight was attached to the consistency of findings among the several studies. However, to simplify presentation, only the combined results are highlighted here.

Of the 1,123,000 men who entered the seven prospective studies and who provided usable histories of smoking habits (and other characteristics such as age), 37,391 men died during the subsequent months or years of the studies. No analyses of data for females from prospective studies are presently available.

To permit ready comparison of the mortality experience of smokers and non-smokers, two concepts are widely used in the studies—excess deaths of smokers compared with non-smokers, and mortality ratio. After adjustments for differences in age and the number of cigarette smokers and non-smokers, an expected number of deaths of smokers is derived on the basis of deaths among non-smokers. Excess deaths are thus the number of actual (observed) deaths among smokers in excess of the number expected. The mortality ratio, for which the method of computation is described in Chapter 8, measures the relative death rates of smokers and non-smokers. If the age-adjusted death rates are the same, the mortality ratio will be 1.0; if the death rates of smokers are double those of non-smokers, the mortality ratio will be 2.0. (Expressed as a percentage, this example would be equivalent to a 100 percent increase.).

Table 2 presents the accumulated and combined data on 14 disease categories for which the mortality ratio of cigarette smokers to non-smokers was 1.5 or greater.

The mortality ratio for male cigarette smokers compared with non-smokers, for all causes of death taken together, is 1.68, representing a total death rate nearly 70 percent higher than for non-smokers. (This ratio includes death rates for diseases not listed in the table as well as for the 14 disease categories shown.)

In the combined results from the seven studies, the mortality ratio of cigarette smokers over non-smokers was particularly high for a number of diseases: cancer of the lung (10.8), bronchitis and emphysema (6.1), can-

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TABLE 2.[1]—*Expected and observed deaths for smokers of cigarettes only and mortality ratios in seven prospective studies*

Underlying cause of death	Expected deaths	Observed deaths	Mortality ratio
Cancer of lung (162-3) [2]	170.3	1,833	10.8
Bronchitis and emphysema (502, 521.1)	89.5	546	6.1
Cancer of larynx (161)	14.0	75	5.4
Oral cancer (140-8)	37.0	152	4.1
Cancer of esophagus (150)	33.7	113	3.4
Stomach and duodenal ulcers (540, 541)	105.1	294	2.8
Other circulatory diseases (451-68)	254.0	649	2.6
Cirrhosis of liver (581)	169.2	379	2.2
Cancer of bladder (181)	111.8	216	1.9
Coronary artery disease (420)	6,430.7	11,177	1.7
Other heart diseases (421-2, 430-4)	526.0	888	1.7
Hypertensive heart (440-3)	409.2	631	1.5
General arteriosclerosis (450)	210.7	310	1.5
Cancer of kidney (180)	79.0	120	1.5
All causes [3]	15,653.9	23,223	1.68

[1] Abridged from Table 26, Chapter 8, Mortality.
[2] International Statistical Classification numbers in parentheses.
[3] Includes all other causes of death as well as those listed above.

cer of the larynx (5.4), oral cancer (4.1), cancer of the esophagus (3.4), peptic ulcer (2.8), and the group of other circulatory diseases (2.6). For coronary artery disease the mortality ratio was 1.7.

Expressed in percentage-form, this is equivalent to a statement that for coronary artery disease, the leading cause of death in this country, the death rate is 70 percent higher for cigarette smokers. For chronic bronchitis and emphysema, which are among the leading causes of severe disability, the death rate for cigarette smokers is 500 percent higher than for non-smokers. For lung cancer, the most frequent site of cancer in men, the death rate is nearly 1,000 percent higher.

Other Findings of the Prospective Studies

In general, the greater the number of cigarettes smoked daily, the higher the death rate. For men who smoke fewer than 10 cigarettes a day, according to the seven prospective studies, the death rate from all causes is about 40 percent higher than for non-smokers. For those who smoke from 10 to 19 cigarettes a day, it is about 70 percent higher than for non-smokers; for those who smoke 20 to 39 a day, 90 percent higher; and for those who smoke 40 or more, it is 120 percent higher.

Cigarette smokers who stopped smoking before enrolling in the seven studies have a death rate about 40 percent higher than non-smokers, as against 70 percent higher for current cigarette smokers. Men who began smoking before age 20 have a substantially higher death rate than those who began after age 25. Compared with non-smokers, the mortality risk of cigarette smokers, after adjustments for differences in age, increases with duration of smoking (number of years), and is higher in those who stopped after age 55 than for those who stopped at an earlier age.

In two studies which recorded the degree of inhalation, the mortality ratio for a given amount of smoking was greater for inhalers than for non-inhalers.

The ratio of the death rates of smokers to that of non-smokers is highest

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at the earlier ages (40–50) represented in these studies, and declines with increasing age.

Possible relationships of death rates and other forms of tobacco use were also investigated in the seven studies. The death rates for men smoking less than 5 cigars a day are about the same as for non-smokers. For men smoking more than 5 cigars daily, death rates are slightly higher. There is some indication that these higher death rates occur primarily in men who have been smoking more than 30 years and who inhale the smoke to some degree. The death rates for pipe smokers are little if at all higher than for non-smokers, even for men who smoke 10 or more pipefuls a day and for men who have smoked pipes more than 30 years.

Excess Mortality

Several of the reports previously published on the prospective studies included a table showing the distribution of the excess number of deaths of cigarette smokers among the principal causes of death. The hazard must be measured not only by the mortality ratio of deaths in smokers and non-smokers, but also by the importance of a particular disease as a cause of death.

In all seven studies, coronary artery disease is the chief contributor to the excess number of deaths of cigarette smokers over non-smokers, with lung cancer uniformly in second place. For all seven studies combined, coronary artery disease (with a mortality ratio of 1.7) accounts for 45 percent of the excess deaths among cigarette smokers, whereas lung cancer (with a ratio of 10.8) accounts for 16 percent.

Some of the other categories of diseases that contribute to the higher death rates for cigarette smokers over non-smokers are diseases of the heart and blood vessels, other than coronary artery disease, 14 percent; cancer sites other than lung, 8 percent; and chronic bronchitis and emphysema, 4 percent.

Since these diseases as a group are responsible for more than 85 percent of the higher death rate among cigarette smokers, they are of particular interest to public health authorities and the medical profession.

Associations and Causality

The array of information from the prospective and retrospective studies of smokers and non-smokers clearly establishes an association between cigarette smoking and substantially higher death rates. The mortality ratios in Table 2 provide an approximate index of the relative strength of this association, for all causes of death and for 14 disease categories.

In this inquiry the epidemiologic method was used extensively in the assessment of causal factors in the relationship of smoking to health among human beings upon whom direct experimentation could not be imposed. Clinical, pathological, and experimental evidence was thoroughly considered and often served to suggest an hypothesis or confirm or contradict other findings. When coupled with the other data, results from the epidemiologic

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studies can provide the basis upon which judgments of causality may be made.

It is recognized that no simple cause-and-effect relationship is likely to exist between a complex product like tobacco smoke and a specific disease in the variable human organism. It is also recognized that often the coexistence of several factors is required for the occurrence of a disease, and that one of the factors may play a determinant role: that is, without it, the other factors (such as genetic susceptibility) seldom lead to the occurrence of the disease.

The Effects of Smoking: Principal Findings

Cigarette smoking is associated with a 70 percent increase in the age-specific death rates of males. The total number of excess deaths causally related to cigarette smoking in the U.S. population cannot be accurately estimated. In view of the continuing and mounting evidence from many sources, it is the judgment of the Committee that cigarette smoking contributes substantially to mortality from certain specific diseases and to the overall death rate.

Lung Cancer

Cigarette smoking is causally related to lung cancer in men: the magnitude of the effect of cigarette smoking far outweighs all other factors. The data for women, though less extensive, point in the same direction.

The risk of developing lung cancer increases with duration of smoking and the number of cigarettes smoked per day, and is diminished by discontinuing smoking. In comparison with non-smokers, average male smokers of cigarettes have approximately a 9- to 10-fold risk of developing lung cancer and heavy smokers at least a 20-fold risk.

The risk of developing cancer of the lung for the combined group of pipe smokers, cigar smokers, and pipe and cigar smokers is greater than for non-smokers, but much less than for cigarette smokers.

Cigarette smoking is much more important than occupational exposures in the causation of lung cancer in the general population.

Chronic Bronchitis and Emphysema

Cigarette smoking is the most important of the causes of chronic bronchitis in the United States, and increases the risk of dying from chronic bronchitis and emphysema. A relationship exists between cigarette smoking and emphysema but it has not been established that the relationship is causal. Studies demonstrate that fatalities from this disease are infrequent among non-smokers.

For the bulk of the population of the United States, the relative importance of cigarette smoking as a cause of chronic broncho-pulmonary disease is much greater than atmospheric pollution or occupational exposures.

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Cardiovascular Diseases

It is established that male cigarette smokers have a higher death rate from coronary artery disease than non-smoking males. Although the causative role of cigarette smoking in deaths from coronary disease is not proven, the Committee considers it more prudent from the public health viewpoint to assume that the established association has causative meaning than to suspend judgment until no uncertainty remains.

Although a causal relationship has not been established, higher mortality of cigarette smokers is associated with many other cardiovascular diseases, including miscellaneous circulatory diseases, other heart diseases, hypertensive heart disease, and general arteriosclerosis.

Other Cancer Sites

Pipe smoking appears to be causally related to lip cancer. Cigarette smoking is a significant factor in the causation of cancer of the larynx. The evidence supports the belief that an association exists between tobacco use and cancer of the esophagus, and between cigarette smoking and cancer of the urinary bladder in men, but the data are not adequate to decide whether these relationships are causal. Data on an association between smoking and cancer of the stomach are contradictory and incomplete.

The Tobacco Habit and Nicotine

The habitual use of tobacco is related primarily to psychological and social drives, reinforced and perpetuated by the pharmacological actions of nicotine.

Social stimulation appears to play a major role in a young person's early and first experiments with smoking. No scientific evidence supports the popular hypothesis that smoking among adolescents is an expression of rebellion against authority. Individual stress appears to be associated more with fluctuations in the amount of smoking than with the prevalence of smoking. The overwhelming evidence indicates that smoking—its beginning, habituation, and occasional discontinuation—is to a very large extent psychologically and socially determined.

Nicotine is rapidly changed in the body to relatively inactive substances with low toxicity. The chronic toxicity of small doses of nicotine is low in experimental animals. These two facts, when taken in conjunction with the low mortality ratios of pipe and cigar smokers, indicate that the chronic toxicity of nicotine in quantities absorbed from smoking and other methods of tobacco use is very low and probably does not represent an important health hazard.

The significant beneficial effects of smoking occur primarily in the area of mental health, and the habit originates in a search for contentment. Since no means of measuring the quantity of these benefits is apparent, the Committee finds no basis for a judgment which would weigh benefits against hazards of smoking as it may apply to the general population.

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The Committee's Judgment in Brief

On the basis of prolonged study and evaluation of many lines of converging evidence, the Committee makes the following judgment:

Cigarette smoking is a health hazard of sufficient importance in the United States to warrant appropriate remedial action.

B. COMMENTS AND DETAILED CONCLUSIONS

(A Guide to Part II of the Report)

All conclusions formally adopted by the Committee are presented at the end of this section in bold-faced type for convenience of reference. In the interest of conciseness, the documentation and most of the discussion are omitted from this condensation. Together with the tables of contents which appear at the beginning of each chapter in Part II, it is intended as a guide to the Report.

Chemistry and Carcinogenicity of Tobacco and Tobacco Smoke

Condensates of tobacco smoke are carcinogenic when tested by application to the skin of mice and rabbits and by subcutaneous injection in rats (Chapter 9, pp. 143–145). Bronchogenic carcinoma has not been produced by the application of tobacco extracts, smoke, or condensates to the lung or the tracheobronchial tree of experimental animals with the possible exception of dogs (Chapter 9, p. 165).

Bronchogenic carcinoma has been produced in laboratory animals by the administration of polycyclic aromatic hydrocarbons, certain metals, radioactive substances, and viruses. The histopathologic characteristics of the tumors produced are similar to those observed in man and are predominantly of the squamous variety (Chapter 9, pp. 166–167).

Seven polycyclic hydrocarbon compounds isolated from cigarette smoke have been established to be carcinogenic in laboratory animals. The results of a number of assays for carcinogenicity of tobacco smoke tars present a puzzling anomaly: the total tar from cigarettes has many times the carcinogenic potency of benzo(a)pyrene present in the tar. The other carcinogens known to be present in tobacco smoke are, with the exception of dibenzo(a,i)pyrene, much less potent than benzo(a)pyrene and they are present in smaller amounts. Apparently, therefore, the whole is greater than the sum of the known parts. This discrepancy may possibly be due to the presence of cocarcinogens in tobacco smoke, and or damage to mucus production and ciliary transport mechanism (Chapter 6, p. 61, Chapter 9, p. 144 and Chapter 10, pp. 267–269).

There is abundant evidence that cancer of the skin can be induced in man by industrial exposure to soots, coal tar, pitch, and mineral oils. All of these

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contain various polycyclic aromatic hydrocarbons proven to be carcinogenic in many species of animals. Some of these hydrocarbons are also present in tobacco smoke. It is reasonable to assume that these can be carcinogenic for man also (Chapter 9, pp. 146–148).

Genetic factors play a significant role in the development of pulmonary adenomas in mice. It is possible that genetic factors can influence the smoking habit and the response in man to carcinogens in smoke. However, there is no evidence that they have played an appreciable role in the great increase of lung cancer in man since the beginning of this century (Chapter 9, p. 190).

Components of the gas phase of cigarette smoke have been shown to produce various undesirable effects on test animals or organs. One of these effects is suppression of ciliary transport activity, an important cleansing function in the trachea and bronchi (Chapter 6, p. 61 and Chapter 10, pp. 267–270).

Characterization of the Tobacco Habit

The habitual use of tobacco is related primarily to psychological and social drives, reinforced and perpetuated by the pharmacological actions of nicotine on the central nervous system. Nicotine-free tobacco or other plant materials do not satisfy the needs of those who acquire the tobacco habit (Chapter 13, p. 354).

The tobacco habit should be characterized as an habituation rather than an addiction. Discontinuation of smoking, although possessing the difficulties attendant upon extinction of any conditioned reflex, is accomplished best by reinforcing factors which interrupt the psychogenic drives. Nicotine substitutes or supplementary medications have not been proven to be of major benefit in breaking the habit (Chapter 13, p. 354).

Pathology and Morphology

Several types of epithelial changes are much more common in the trachea and bronchi of cigarette smokers, with or without lung cancer, than of non-smokers and of patients without lung cancer. These epithelial changes are (a) loss of cilia, (b) basal cell hyperplasia, and (c) appearance of atypical cells with irregular hyperchromatic nuclei. The degree of each of the epithelial changes in general increases with the number of cigarettes smoked. Extensive atypical changes have been seen most frequently in men who smoked two or more packs of cigarettes a day.

Women cigarette smokers, in general, have the same epithelial changes as men smokers. However, at given levels of cigarette use, women appear to show fewer atypical cells than do men. Older men smokers have more atypical cells than younger men smokers. Men who smoke either pipes or cigars have more epithelial changes than non-smokers, but have fewer changes than cigarette smokers consuming approximately the same amount of tobacco. Male ex-cigarette smokers have less hyperplasia and fewer atypical cells than current cigarette smokers.

It may be concluded, on the basis of human and experimental evidence, that some of the advanced epithelial hyperplastic lesions with many atypical

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cells, as seen in the bronchi of cigarette smokers, are probably premalignant (Chapter 9, pp. 167–173).

Typing of Tumors.—Squamous and oval-cell carcinomas (Group I of Kreyberg's classification) comprise the predominant types associated with the increase of lung cancer in the male population. In several studies, adenocarcinomas (Group II) have also shown a definite increase, although to a much lesser degree. The histological typing of lung cancer is reliable, but the use of the ratio of histological types as an index of the magnitude of increase in lung cancer is of limited value (Chapter 9, pp. 173–175).

Functional and Pathological Changes.—Cigarette smoke produces significant funtional alterations in the trachea, bronchus, and lung. Like several other agents, cigarette smoke can reduce or abolish ciliary motility in experimental animals. Postmortem examination of bronchi from smokers shows a decrease in the number of ciliated cells, shortening of the remaining cilia, and changes in goblet cells and mucous glands. The implication of these morphological observations is that functional impairment would result.

In animal experiments, cigarette smoke appears to affect the physical characteristics of the lung-lining layer and to impair alveolar (air sac) stability. Alveolar phagocytes ingest tobacco smoke components and assist in their removal from the lung. This phagocytic clearance mechanism breaks down under the stress of protracted high-level exposure to cigarette smoke, and smoke components accumulate in the lungs of experimental animals (Chapter 10, pp. 269–270).

The chronic effects of cigarette smoking upon pulmonary function are manifested mainly by a reduction in ventilatory function as measured by the forced expiratory volume (Chapter 10, pp. 289–292).

Histopathological alterations occur as a result of tobacco smoke exposure in the tracheobronchial tree and in the lung parenchyma of man. Changes regularly found in chronic bronchitis—increase in the number of goblet cells, and hypertrophy and hyperplasia of bronchial mucous glands—are more often present in the bronchi of smokers than non-smokers. Cigarette smoke produces significant functional alterations in the upper and lower airways to the lungs. Such alterations could be expected to interfere with the cleansing mechanisms of the lung.

Pathological changes in pulmonary parenchyma, such as rupture of alveolar septa (partitions of the air sacs) and fibrosis, have a remarkably close association with past history of cigarette smoking. These latter changes cannot be related with certainty to emphysema or other recognized diseases at the present time (Chapter 10, pp. 270–275).

Mortality

The death rate for smokers of cigarettes only, who were smoking at the time of entry into the particular prospective study, is about 70 percent higher than that for non-smokers. The death rates increase with the amount smoked. For groups of men smoking less than 10, 10–19, 20–39, and 40 cigarettes and over per day, respectively, the death rates are about 40 percent, 70 per-

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cent, 90 percent, and 120 percent higher than for non-smokers. The ratio of the death rates of smokers to non-smokers is highest at the earlier ages (40–50) represented in these studies, and declines with increasing age. The same effect appears to hold for the ratio of the death rate of heavy smokers to that of light smokers. In the studies that provided this information, the mortality ratio of cigarette smokers to non-smokers was substantially higher for men who started to smoke under age 20 than for men who started after age 25. The mortality ratio was increased as the number of years of smoking increased. In two studies which recorded the degree of inhalation, the mortality ratio for a given amount of smoking was greater for inhalers than for non-inhalers. Cigarette smokers who had stopped smoking prior to enrollment in the study had mortality ratios about 1.4 as against 1.7 for current cigarette smokers. The mortality ratio of ex-cigarette smokers increased with the number of years of smoking and was higher for those who stopped after age 55 than for those who stopped at an earlier age (Chapter 8, p. 93).

The biases from non-response and from errors of measurement that are difficult to avoid in mass studies may have resulted in some over-estimation of the true mortality ratios for the complete populations. In our judgment, however, such biases can account for only a part of the elevation in mortality ratios found for cigarette smokers (Chapter 8, p. 96).

Death rates of cigar smokers are about the same as those of non-smokers for men smoking less than five cigars daily. For men smoking five or more cigars daily, death rates were slightly higher (9 percent to 27 percent) than for non-smokers in the four studies that gave this information. There is some indication that this higher death rate occurs primarily in men who have been smoking for more than 30 years and in men who stated that they inhaled the smoke to some degree. Death rates for current pipe smokers were little if at all higher than for non-smokers, even with men smoking 10 or more pipefuls per day and with men who had smoked pipes for more than 30 years. Ex-cigar and ex-pipe smokers, on the other hand, showed higher death rates than both non-smokers and current pipe or cigar smokers in four out of five studies (Chapter 8, p. 94). The explanation is not clear but may be that a substantial number of such smokers stopped because of illness.

Mortality by Cause of Death.—In the combined results from the seven prospective studies, the mortality ratio of cigarette smokers was particularly high for a number of diseases. There is a further group of diseases, including some of the most important chronic diseases, for which the mortality ratio for cigarette smokers lay between 1.2 and 2.0. The explanation of the moderate elevations in mortality ratios in this large group of causes is not clear. Part may be due to the sources of bias previously mentioned or to some constitutional and genetic difference between cigarette smokers and non-smokers. There is also the possibility that cigarette smoking has some general debilitating effect, although no medical evidence that clearly supports this hypothesis can be cited (Chapter 8, p. 105).

In all seven studies, coronary artery disease is the chief contributor to the excess number of deaths of cigarette smokers over non-smokers, with lung cancer uniformly in second place (Chapter 8, p. 108).

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For cigar and pipe smokers combined, there was a suggestion of high mortality ratios for cancers of the mouth, esophagus, larynx and lung, and for stomach and duodenal ulcers. These ratios are, however, based on small numbers of deaths (Chapter 8, p. 107).

Cancer by Site

Lung Cancer

Cigarette smoking is causally related to lung cancer in men; the magnitude of the effect of cigarette smoking far outweighs all other factors. The data for women, though less extensive, point in the same direction.

The risk of developing lung cancer increases with duration of smoking and the number of cigarettes smoked per day, and is diminished by discontinuing smoking.

The risk of developing cancer of the lung for the combined group of pipe smokers, cigar smokers, and pipe and cigar smokers, is greater than for non-smokers, but much less than for cigarette smokers. The data are insufficient to warrant a conclusion for each group individually (Chapter 9, p. 196).

Oral Cancer

The causal relationship of the smoking of pipes to the development of cancer of the lip appears to be established.

Although there are suggestions of relationships between cancer of other specific sites of the oral cavity and the several forms of tobacco use, their causal implications cannot at present be stated (Chapter 9, pp. 204–205).

Cancer of the Larynx

Evaluation of the evidence leads to the judgment that cigarette smoking is a significant factor in the causation of laryngeal cancer in the male (Chapter 9, p. 212).

Cancer of the Esophagus

The evidence on the tobacco-esophageal cancer relationship supports the belief that an association exists. However, the data are not adequate to decide whether the relationship is causal (Chapter 9, p. 218).

Cancer of the Urinary Bladder

Available data suggest an association between cigarette smoking and urinary bladder cancer in the male but are not sufficient to support a judgment on the causal significance of this association (Chapter 9, p. 225).

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Stomach Cancer

No relationship has been established between tobacco use and stomach cancer (Chapter 9, p. 229).

Non-Neoplastic Respiratory Diseases, Particularly Chronic Bronchitis and Pulmonary Emphysema

Cigarette smoking is the most important of the causes of chronic bronchitis in the United States, and increases the risk of dying from chronic bronchitis.

A relationship exists between pulmonary emphysema and cigarette smoking but it has not been established that the relationship is causal. The smoking of cigarettes is associated with an increased risk of dying from pulmonary emphysema.

For the bulk of the population of the United States, the importance of cigarette smoking as a cause of chronic bronchopulmonary disease is much greater than that of atmospheric pollution or occupational exposures.

Cough, sputum production, or the two combined are consistently more frequent among cigarette smokers than among non-smokers.

Cigarette smoking is associated with a reduction in ventilatory function. Among males, cigarette smokers have a greater prevalence of breathlessness than non-smokers.

Cigarette smoking does not appear to cause asthma.

Although death certification shows that cigarette smokers have a moderately increased risk of death from influenza and pneumonia, an association of cigarette smoking and infectious diseases is not otherwise substantiated (Chapter 10, p. 302).

Cardiovascular Disease

Smoking and nicotine administration cause acute cardiovascular effects similar to those induced by stimulation of the autonomic nervous system, but these effects do not account well for the observed association between cigarette smoking and coronary disease. It is established that male cigarette smokers have a higher death rate from coronary disease than non-smoking males. The association of smoking with other cardiovascular disorders is less well established. If cigarette smoking actually caused the higher death rate from coronary disease, it would on this account be responsible for many deaths of middle-aged and elderly males in the United States. Other factors such as high blood pressure, high serum cholesterol, and excessive obesity are also known to be associated with an unusually high death rate from coronary disease. The causative role of these factors in coronary disease, though not proven, is suspected strongly enough to be a major reason for taking countermeasures against them. It is also more prudent to assume that the established association between cigarette smoking and coro-

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nary disease has causative meaning than to suspend judgment until no uncertainty remains (Chapter 11, p. 327).

Male cigarette smokers have a higher death rate from coronary artery disease than non-smoking males, but it is not clear that the association has causal significance.

Other Conditions

Peptic Ulcer

Epidemiological studies indicate an association between cigarette smoking and peptic ulcer which is greater for gastric than for duodenal ulcer (Chapter 12, p. 340).

Tobacco Amblyopia

Tobacco amblyopia (dimness of vision unexplained by an organic lesion) has been related to pipe and cigar smoking by clinical impressions. The association has not been substantiated by epidemiological or experimental studies (Chapter 12, p. 342).

Cirrhosis of the Liver

Increased mortality of smokers from cirrhosis of the liver has been shown in the prospective studies. The data are not sufficient to support a direct or causal association (Chapter 12, p. 342).

Maternal Smoking and Infant Birth Weight

Women who smoke cigarettes during pregnancy tend to have babies of lower birth weight.

Information is lacking on the mechanism by which this decrease in birth weight is produced.

It is not known whether this decrease in birth weight has any influence on the biological fitness of the newborn (Chapter 12, p. 343).

Smoking and Accidents

Smoking is associated with accidental deaths from fires in the home.

No conclusive information is available on the effects of smoking on traffic accidents (Chapter 12, p. 345).

Morphological Constitution of Smokers

The available evidence suggests the existence of some morphological differences between smokers and non-smokers, but is too meager to permit a conclusion (Chapter 15, p. 387).

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A clear cut smoker's personality has not emerged from the results so far published. While smokers differ from non-smokers in a variety of characteristics, none of the studies has shown a single variable which is found solely in one group and is completely absent in another. Nor has any single variable been verified in a sufficiently large proportion of smokers and in sufficiently few non-smokers to consider it an "essential" aspect of smoking.

The overwhelming evidence points to the conclusion that smoking—its beginning, habituation, and occasional discontinuation—is to a large extent psychologically and socially determined. This does not rule out physiological factors, especially in respect to habituation, nor the existence of predisposing constitutional or hereditary factors (Chapter 14, p. 377).

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From Vital and Health Statistics of the National Center for Health Statistics Number 154 • May 27, 1988

Relationships Between Smoking and Other Unhealthy Habits: United States, 1985

by Charlotte A. Schoenborn, M.P.H., and Veronica Benson, Division of Health Interview Statistics

Introduction

Smoking has been identified as the number one cause of preventable death in the United States (Office on Smoking and Health, 1979). Extensive medical research has clearly documented the deleterious health effects of smoking. Additional evidence has indicated that combining smoking with other unhealthy behaviors can multiply the probability of serious illness; for instance, combining smoking and heavy drinking greatly increases the probability of oral cancer (Office on Smoking and Health, 1979). Although exploring possible effects for health outcomes of combining smoking with other bad habits is beyond the scope of this report, the following analysis can answer the question of whether smokers have other unhealthy habits that may, based on other available evidence (Belloc and Breslow, 1972; Wiley and Camacho, 1980), increase their probability of succumbing to serious illness, disability, and even death.

Earlier research efforts have examined smoking behavior in relation to specific behaviors such as alcohol and caffeine use (Istvan and Matarazzo, 1984), food consumption (Grunberg and Morse, 1984), body weight (Albanes, Jones, Micozzi, and Mattson, 1987), and exercise (Faulkner, Bailey, and Mirwald, 1987; Salonen et al., 1987). Other investigators have studied interrelationships among a wider variety of behaviors (Blair, Jacobs, and Powell, 1985; Langlie, 1979; Mechanic and Cleary, 1980; Norman, 1985).

This report presents national data on the interrelationships between smoking and eight other behaviors judged to be unhealthy based on currently available evidence. Cross-sectional and longitudinal studies performed in Alameda County, California, since the mid-1960's found that certain health behaviors were related to subsequent morbidity and mortality (Belloc and Breslow, 1972; Breslow and Enstrom, 1980; Wiley and Camacho, 1980). The behaviors studied in Alameda County were smoking, alcohol consumption, hours of sleep, exercise, eating breakfast, snacking, and being overweight. This report follows the Alameda model, including behaviors that, although not measured identically, are conceptually similar to those shown in the Alameda study to be related to health status and survival.

A similar report using data from the 1983 National Health Interview Survey (NHIS) Alcohol and Health Practices Questionnaire has also been prepared (Hendershot and Bloom, unpublished). Information such as this may aid health educators in planning more effective smoking cessation programs. The case of alcohol and smoking is illustrative. Evidence suggests that a substantial proportion of smokers are heavier drinkers, according to criteria established by the National Institute on Alcohol Abuse and Alcoholism (Hendershot and Bloom, unpublished). It might be hypothesized that, at least for some portion of the smoking population, alcohol consumption may interfere with attempts to quit smoking by reducing the smoker's resolve to quit smoking. If this premise is correct, health promotion efforts aimed at smokers could benefit from simultaneously promoting reduction (or elimination) of alcohol consumption. Similarly, smokers have been found to be less likely to exercise (Hendershot and Bloom, unpublished). Health educators might consider the possibility that increasing smokers' participation in sports activities might stimulate physiological or psychological responses that would enhance the desire to quit smoking. Although reasons for associations between smoking and other health practices are not well understood, it is important to consider such associations when designing programs to change behavior. The following analysis illuminates a number of behaviors other than smoking that could receive attention in lifestyle-oriented smoking cessation programs.

Methods

This report is based on data from the 1985 National Health Interview Survey of Health Promotion and Disease

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Prevention (NHIS-HPDP). The NHIS is a continuous, nationwide, household interview survey of the civilian noninstitutionalized population of the United States, conducted by the National Center for Health Statistics. The NHIS consists of two parts: (a) a basic health and demographic questionnaire that remains the same from year to year and (b) special topic questionnaires that change from year to year. In 1985 the special topic section was devoted entirely to health promotion topics. A detailed description of the survey and its methods is available (NCHS, 1986, 1988).

The sample for the NHIS-HPDP consisted of one randomly selected adult per family, aged 18 years or over. Self-response was required. HPDP questionnaires were completed for 33,630 persons, representing an estimated 90 percent of eligible respondents. This analysis is limited to the 32,517 persons aged 20 years and over.

Table 1 shows the percent of current smokers, former smokers, and never smokers who had each of the eight unhealthy behaviors: sleeping six hours or less, skipping breakfast, snacking daily, being less physically active than other per-

Table 1. Percent of persons 20 years of age and over who had selected unhealthy behaviors by smoking status, number of cigarettes smoked daily, health practices, and sex: United States, 1985

[Data are based on household interviews of the civilian noninstitutionalized population. The survey design, general qualifications, and information on the reliability of the estimates are given in the Technical notes]

Health practices and sex	Smoking status				Number of cigarettes smoked daily			
	All smoking statuses	Never smoker	Former smoker	Current smoker	Less than 15	15–24	25–34	35 or more
	Percent							
Sleeps 6 hours or less								
Both sexes	22.3	20.9	20.9	25.3	22.6	23.9	27.9	33.1
Male	22.9	21.5	21.3	26.0	23.8	23.8	28.0	33.1
Female	21.7	20.6	20.3	24.5	21.7	24.1	27.8	34.0
Never eats breakfast								
Both sexes	24.3	18.3	19.0	37.6	28.0	38.0	45.7	50.6
Male	25.3	20.8	19.2	35.6	25.0	34.3	43.4	47.9
Female	23.5	16.8	18.5	39.6	30.3	41.6	48.8	55.6
Snacks daily								
Both sexes	38.4	38.2	40.3	37.5	34.3	37.4	41.3	41.7
Male	39.8	40.7	39.6	39.2	35.5	39.4	42.9	41.0
Female	37.2	36.8	41.3	35.8	33.5	35.4	38.9	43.0
Less physically active[2]								
Both sexes	18.9	17.3	18.1	21.8	21.0	21.3	23.1	24.0
Male	15.6	13.5	15.1	18.5	17.3	17.7	19.7	21.1
Female	21.8	19.6	22.7	25.2	23.8	24.9	27.9	29.5
Sedentary[3]								
Both sexes	56.7	55.3	54.3	58.9	54.5	59.8	60.8	63.4
Male	50.5	43.2	50.7	55.5	50.4	55.6	57.4	60.6
Female	62.3	62.3	59.7	62.4	57.6	64.1	65.6	68.9
Overweight[4]								
Both sexes	24.6	25.3	29.0	20.1	18.9	17.7	22.6	26.9
Male	26.7	26.1	32.2	22.0	18.8	19.1	24.9	29.9
Female	22.8	24.9	24.2	18.1	18.9	16.4	19.2	21.1
Heavier drinker[5]								
Both sexes	7.8	3.6	9.0	12.9	10.0	10.9	16.9	22.0
Male	13.2	7.9	12.3	19.6	16.3	16.7	22.6	28.8
Female	3.0	1.1	3.9	5.9	5.3	5.1	8.9	8.7
Consumes 5 drinks or more[6]								
Both sexes	12.1	7.2	11.9	20.1	15.4	20.6	24.0	26.7
Male	20.9	16.1	17.0	30.9	26.1	32.2	33.3	33.9
Female	4.4	2.1	4.4	9.1	7.6	9.1	11.2	13.1

[1] Current smokers only.
[2] Based on perceived level of physical activity.
[3] Sedentary equals energy expenditure on leisure activity of 0.0–1.4 kilocalories/kilogram/day.
[4] 20 percent or more above desirable weight, based on 1983 Metropolitan Life Insurance Company standards (1983, *Statist. Bull.* 64(1):2–9). National Health Interview Survey data are self-reported and estimates may vary from those that would be obtained if physical measurements were taken.
[5] Measure developed by the National Institute on Alcohol Abuse and Alcoholism. Categories based on ounces of ethanol consumed during the past 2 weeks. The drinker is defined as having had an average 1.0 ounce of ethanol (2 drinks) or more per day.
[6] 5 drinks or more on 10 days or more in the past year.

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sons of the same age, being sedentary in terms of leisure time sports activities, being significantly overweight (20 percent or more), drinking heavily (an average of two drinks or more daily), and having five drinks or more on 10 days or more in the past year. Table 2 shows the same relationships as those shown in table 1, after adjusting for differences in the age distributions in the various smoking status groups. Because health behaviors vary substantially by age, it is important to rule out age as the explanation for observed differences among groups with different smoking patterns. For this reason, discussion of results will be limited to the age-adjusted table (table 2). It should be noted, however, that the actual prevalence levels of behaviors are the unadjusted figures in table 1.

In this report, terms such as "similar" and "no difference" mean that there is no statistically significant difference between the measures being compared. Terms relating to difference (for example, "greater than" or "less than") indicate that differences are statistically significant. The *t*-test, with a

Table 2. Age-adjusted percent of persons 20 years of age and over who had selected unhealthy behaviors by smoking status, number of cigarettes smoked daily, health practices, and sex: United States, 1985

(Data are based on household interviews of the civilian noninstitutionalized population. The survey design, general qualifications, and information on the reliability of the estimates are given in the Technical notes)

Health practices and sex	Smoking status				Number of cigarettes smoked daily[1]			
	All smoking statuses	Never smoker	Former smoker	Current smoker	Less than 15	15–24	25–34	35 or more
	Age-adjusted percent							
Sleeps 6 hours or less								
Both sexes	22.3	21.0	21.2	24.6	22.6	23.3	27.1	31.6
Male	22.8	21.5	22.5	24.9	23.7	22.5	26.0	31.1
Female	21.6	20.4	19.9	24.4	21.8	23.9	28.6	34.1
Never eats breakfast								
Both sexes	24.4	18.1	21.2	35.4	26.7	35.9	42.1	47.6
Male	24.8	18.9	22.3	33.3	23.4	31.9	39.4	45.5
Female	24.0	17.7	19.8	37.6	29.2	39.8	45.9	51.7
Snacks daily								
Both sexes	38.4	38.4	40.8	36.9	33.6	36.8	40.4	42.4
Male	39.6	39.3	40.4	38.5	34.7	38.2	42.3	42.5
Female	37.4	37.6	41.5	35.3	32.7	35.2	37.7	42.5
Less physically active[2]								
Both sexes	18.9	17.2	18.4	21.8	20.8	21.0	23.1	26.1
Male	15.7	13.2	14.6	18.8	18.0	17.7	19.0	23.6
Female	21.9	19.9	23.3	24.9	23.0	24.5	29.9	30.9
Sedentary[3]								
Both sexes	56.7	55.6	52.3	60.6	57.0	61.7	64.1	64.7
Male	51.0	46.6	47.7	57.2	53.0	57.6	60.5	62.1
Female	61.8	61.1	58.5	64.3	60.0	65.7	68.8	70.5
Overweight[4]								
Both sexes	24.6	26.4	27.0	19.6	19.3	17.4	22.2	22.4
Male	26.5	28.1	30.0	21.2	19.4	18.7	23.0	27.2
Female	22.7	24.9	23.0	17.9	19.3	16.0	20.5	19.7
Heavier drinker[5]								
Both sexes	7.8	3.6	8.9	12.6	9.5	10.7	16.9	22.5
Male	13.1	7.9	12.7	18.9	15.3	15.5	21.8	28.9
Female	3.0	1.1	3.7	6.1	5.2	5.7	10.2	9.5
Consumes 5 drinks or more[6]								
Both sexes	12.1	6.8	14.1	18.7	13.6	19.0	22.1	26.2
Male	20.5	13.8	21.2	28.7	23.0	29.3	30.6	33.0
Female	4.6	2.2	5.0	8.5	6.7	8.7	10.4	13.2

[1] Current smokers only.
[2] Based on perceived level of physical activity.
[3] Sedentary equals energy expenditure on leisure activity of 0.0–1.4 kilocalories/kilogram/day.
[4] 20 percent or more above desirable weight, based on 1983 Metropolitan Life Insurance Company standards (1983 *Statist. Bull.* 64(1):2–9). National Health Interview Survey data are self-reported, and estimates may vary from those that would be obtained if physical measurements were taken.
[5] Measure developed by the National Institute on Alcohol Abuse and Alcoholism. Category is based on ounces of ethanol consumed during the past 2 weeks; heavier drinker is defined as having had an average 1.0 ounce of ethanol (2 drinks) or more per day.
[6] 5 drinks or more on 10 days or more in the past year.

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critical value of 1.96 (0.05 level of significance), was used to test all comparisons. Lack of comment regarding the difference between any two statistics does not mean the difference was tested and found to be not significant.

Definition of terms

Smoking

Current smokers are defined as persons who had smoked at least 100 cigarettes in their lifetime and reported that they were smoking at the time of interview. Former smokers had smoked 100 cigarettes in their lifetime but were not currently smoking. Never smokers had never smoked 100 cigarettes. Unless otherwise indicated, the term "smokers" in this report refers to current smokers.

Sleep

Sleeping 6 hours or less a night has been defined as an unhealthy habit by other researchers (Belloc and Breslow, 1972; Wiley and Camacho, 1980). It was selected for this analysis on that basis.

Breakfast and snacking

Skipping breakfast and snacking regularly have been identified elsewhere as poor eating habits (Belloc and Breslow, 1972). Breakfast and snacks were respondent-defined in the NHIS-HPDP, and frequency was reported as almost every day, sometimes, rarely, or never. In the interest of brevity for discussion of these behaviors, "almost every day" is abbreviated to "every day," and "rarely or never" is shortened to "never." The terms "skipping breakfast" and "never eating breakfast" are used interchangeably.

Physical activity

Two measures of physical activity were included. The first, physical activity level relative to other persons one's own age, is a subjective indicator, combining an assessment of one's own activity level with an assessment of the activity level of one's contemporaries. This indicator offers comparability with an earlier survey (Hendershot and Bloom, unpublished). The second measure of physical activity is based on a more rigorous definition of exercise levels. Respondents were asked to report their participation in 23 leisure time physical activities (such as walking, jogging, gardening, aerobics, golf, and tennis) over the past 2 weeks. Their participation was converted into total kilocalories of energy expended over a 2-week period and averaged over the 14 days to obtain a daily energy expenditure. Persons with kilocalorie levels of under 1.5 per day were defined as sedentary for this analysis. A more complete description of this methodology has been published previously (Schoenborn, 1986).

Overweight

Overweight is defined as being 20 percent or more above desirable body weight according to the 1983 Metropolitan Life Insurance Company height and weight tables (Metropolitan Life Insurance Company, 1983). All height and weight data in the NHIS-HPDP were self-reported. Further information on this variable is available in the Technical notes.

Heavier drinker

The National Institute on Alcohol Abuse and Alcoholism has defined "heavier drinker" as a person consuming an average of 1.0 ounce or more of ethanol per day; that is, about 2 average-sized drinks of beer, wine, or liquor. The National Institute on Alcohol Abuse and Alcoholism definition was used for classifying persons in this analysis. Information was collected on the frequency of alcohol consumption over the 2 weeks preceding the interview and the average quantity consumed per occasion. These data were then converted into ounces of ethanol using a factor of 0.5 ounce per drink. The number of ounces were summed and then averaged over the 14-day period to obtain an average daily amount consumed.

Five drinks or more

Because health-related patterns in drinking behavior may be masked by using an average daily alcohol consumption (for instance, a person with an average daily alcohol consumption of 2 drinks per day could have consumed 28 drinks in a weekend or 2 drinks a day for 14 days over any given 2-week period), an indicator of repeated heavy drinking episodes was also employed. Respondents were asked on how many days in the past year they had had five drinks or more. For this report, the cutoff of 10 days or more was selected because it suggests that this drinking involved more than holiday-type drinking.

Findings

Sleep

Table 2 shows that, controlling for differences in age composition, about 22 percent of U.S. adults slept an average of 6 hours or less each night. Current smokers were more likely than both former and never smokers to sleep this little. In 1985, approximately one-fourth of current smokers slept 6 hours or less compared with about 21 percent of former smokers and persons who had never smoked. The association between current smoking and getting less sleep was found for men and women. Male current smokers were more likely to sleep 6 hours or less (24.9) than males who had never smoked (21.5). Similarly, female current smokers were more likely to sleep 6 hours or less (24.4) than women who had never smoked and women who had quit smoking—about 20 percent for the latter two groups.

Among current smokers, sleeping 6 hours or less was related to the number of cigarettes smoked. Almost one-third of smokers in the heaviest smoking category (those smoking 35 cigarettes or more daily) slept 6 hours or less compared with 27.1 percent of those smoking 25–34 cigarettes, 23.3 percent of those smoking 15–24 cigarettes, and 22.6 percent of those smoking fewer than 15 cigarettes per day. This dose-response relationship between number of cigarettes smoked and sleeping habits was similar for men and women. Women who smoked 35 cigarettes or more daily were the most likely to sleep 6 hours or less of any of the groups shown in table 2 (34.1 perc

Skipping breakfast

Table 2 shows that, controlling for differences in age composition, skipping breakfast was more prevalent among smokers

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than nonsmokers. On average, more than one-third of current smokers never ate breakfast compared with 21.2 percent of former smokers and 18.1 percent of never smokers. The relationship between cigarette smoking and skipping breakfast was found for both men and women. Male current smokers were more likely to skip breakfast (33.3) than male former smokers (22.3) and male never smokers (18.9). Female current smokers also were more likely to skip breakfast (37.6) than female former smokers (19.8) and female never smokers (17.7). Female smokers were more likely than male smokers to never eat breakfast 37.6 and 33.3, respectively).

Among smokers, skipping breakfast was associated with the amount smoked, the highest prevalence being among those smoking a greater number of cigarettes. Persons smoking 35 cigarettes or more daily were more likely to never eat breakfast (47.6) than persons smoking 25–34 cigarettes (42.1), 15–24 cigarettes (35.9), and less than 15 cigarettes daily (26.7). As was the case with unhealthy sleeping habits, female heavy smokers (35 cigarettes or more daily) were the most likely to have unhealthy breakfast habits of any group shown. About 52 percent of female smokers smoking 35 cigarettes or more daily never ate breakfast compared with 39.8 percent of females smoking 15–24 cigarettes and 29.2 percent of females smoking less than 15 cigarettes daily. A similar relationship between breakfast habits and number of cigarettes smoked was found for men. Male smokers who smoked 35 or more cigarettes daily were more likely to skip breakfast (45.5) than males smoking 25–34 cigarettes (39.4), males smoking 15–24 cigarettes (31.9), and males smoking less than 15 cigarettes daily (23.4).

Snacking

Table 2 shows that, controlling for differences in age composition, about 37 percent of current smokers snacked every day compared with 40.8 percent of former smokers and 38.4 percent of never smokers. On average, about 35 percent of female current smokers snacked daily compared with 41.5 percent of female former smokers and 37.6 percent of female never smokers. For males, snacking habits of smokers versus nonsmokers were not significantly different.

Although smokers on the whole tended to be less likely to snack every day than nonsmokers, heavier smokers were *more* likely to snack. About 42 percent of persons who smoked 35 cigarettes or more daily snacked every day compared with 36.8 percent of those who smoked 15–24 cigarettes daily, 33.6 percent of those who smoked less than 15 cigarettes daily, and 38 percent of those who had never smoked. Males smoking 35 cigarettes or more daily were more likely to snack every day (42.5 percent) than males smoking less than 15 cigarettes daily (34.7 percent). Females who smoked 35 cigarettes or more daily were more likely to snack every day (42.5 percent) than females smoking 15–24 cigarettes daily (35.2 percent) and females smoking less than 15 cigarettes daily (32.7 percent).

Less physically active

Table 2 shows that, controlling for differences in age composition, the perception of being less physically active was more prevalent among current smokers than former and never smokers. About 22 percent of current smokers perceived themselves to be less physically active than their contemporaries compared with 18.4 percent of former smokers and 17.2 percent of never smokers. About 19 percent of male current smokers reported being less physically active than their contemporaries compared with about 15 percent of male former smokers and about 13 percent of male never smokers. About 25 percent of female current smokers reported being less physically active than their contemporaries compared with about 20 percent of female never smokers.

Compared with lighter smokers, heavier smokers tended to be less physically active than their contemporaries. About 26 percent of those smoking 35 or more cigarettes daily were less physically active than their contemporaries, compared with about 21 percent of smokers smoking less than 25 cigarettes daily. About 24 percent of males smoking 35 cigarettes or more daily were less physically active than their contemporaries, compared with 18.0 percent of males smoking less than 15 cigarettes daily. Similarly, among females smoking 35 cigarettes or more daily, about 31 percent reported being less physically active than their contemporaries, compared with 23 percent of females smoking less than 15 cigarettes daily. Women were more likely than men to report being less physically active than others the same age, across all smoking statuses and all smoking levels.

Sedentary activity level

Table 2 shows that, controlling for differences in age composition, current smokers were more likely to be sedentary in terms of leisure time sports activities (60.6 percent) compared with former smokers (52.3 percent) and never smokers (55.6 percent). Male current smokers were more likely to be sedentary (57.2 percent) than male former smokers (47.7 percent) and male never smokers (46.6 percent); the difference between former and never smokers was not statistically significant. As with males, female current smokers were more likely to be sedentary (64 percent) than were female nonsmokers. In contrast to the findings for males, female former smokers were somewhat less likely to be sedentary (58.5 percent) than females who had never smoked (61.1 percent).

For smokers, sedentary behavior also was associated with the number of cigarettes smoked. About 65 percent of those smoking 35 cigarettes or more daily were sedentary compared with 57 percent of those smoking less than 15 cigarettes daily. This association was found for males and females. Males smoking 35 cigarettes or more daily were more likely to be sedentary (62.1 percent) than males smoking 15–24 cigarettes daily (57.6 percent) and males smoking less than 15 cigarettes daily (53.0 percent). Females smoking 35 cigarettes or more daily were more likely to be sedentary (70.5 percent) than females smoking less than 15 cigarettes daily (60.0 percent). Females were more likely than males to be sedentary, across all smoking statuses and regardless of the number of cigarettes smoked.

Overweight

Table 2 shows that, controlling for differences in age composition, current smokers were less likely to be significantly

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overweight (20 percent or more above desirable body weight) than former and never smokers. About 20 percent of current smokers were significantly overweight compared with 27 percent of former smokers and 26.4 percent of never smokers. Male current smokers were less likely to be overweight (21.2 percent) than male former smokers (30.0 percent) and male never smokers (28.1 percent). Similarly, female current smokers were less likely to be overweight (17.9 percent) than female former smokers (23.0 percent) and female never smokers (24.9 percent).

Among male smokers, overweight was related to the number of cigarettes smoked daily, but not in the expected direction. Although smokers on the whole were *less* likely to be overweight, the heaviest smokers were the *most* likely of the smokers to be overweight. Prevalence of overweight among males smoking 35 cigarettes or more daily (27.2 percent) was similar to that of males who had never smoked (28.1 percent). Overweight was substantially less prevalent among males smoking fewer than 25 cigarettes daily (about 19 percent). No clear pattern emerged for female smokers in the relationship between number of cigarettes smoked daily and prevalence of overweight.

Heavier drinker

Table 2 shows that, controlling for differences in age composition, current smokers were more likely than former and never smokers to be heavier drinkers. About 13 percent of current smokers were heavier drinkers compared with 8.9 percent of former smokers and 3.6 percent of never smokers. About 19 percent of male current smokers were heavier drinkers compared with 12.7 percent of male former smokers and 7.9 percent of male never smokers. Female current smokers were more likely to be heavier drinkers (6.1 percent) than female former smokers (3.7 percent) and female never smokers (1.1 percent). Males were more likely than females to be heavier drinkers regardless of smoking status.

Heavier smoking was associated with heavier drinking in the expected direction. Current smokers smoking 35 cigarettes or more daily were more likely to be heavier drinkers (22.5 percent) than those smoking 25–34 cigarettes daily (16.9 percent), 15–24 cigarettes daily (10.7 percent), and less than 15 cigarettes daily (9.5 percent). The dose-response relationship was clearest for males. Among males smoking 35 cigarettes or more daily, 28.9 percent were heavier drinkers compared with 21.8 percent of males smoking 25–34 cigarettes daily, 15.5 percent of males smoking 15–24 cigarettes daily, and 15.3 percent of those smoking less than 15 cigarettes daily. Among females, the relationship between number of cigarettes smoked and heavier drinking habits was dichotomous: About 10 percent of females who smoked 25 cigarettes or more daily were heavier drinkers compared with 5–6 percent of females who smoked fewer than 25 cigarettes. Males were substantially more likely than females to be heavier drinkers, regardless of smoking status or amount smoked.

Five drinks or more

Table 2 shows that, controlling for differences in age composition, current smokers were more likely to have had five drinks or more on 10 days or more during the past year than either former or never smokers. About 19 percent of current smokers had exhibited this drinking behavior compared with 14.1 percent of former smokers and 6.8 percent of never smokers. Male smokers were more likely to have had five drinks or more this many times (28.7 percent) than male former smokers (21.2 percent) and male never smokers (13.8 percent). As with males, female current smokers were more likely to report this behavior (8.5 percent) than were female former smokers (5.0 percent) or never smokers (2.2 percent). Consuming five drinks or more at least 10 times in the past year was more common among males than females across all smoking statuses.

Having five drinks or more on 10 days or more during the past year was also related to the amount smoked. Current smokers smoking 35 cigarettes or more daily were more likely to report this behavior (26.2 percent) than those smoking 25–34 cigarettes (22.1 percent), 15–24 cigarettes (19.0 percent), and less than 15 cigarettes daily (13.6 percent). Males smoking 35 cigarettes or more daily were 10 percentage points more likely to have had five drinks or more at least 10 times in the past year (33.0 percent) than males smoking less than 15 cigarettes daily (23.0 percent). Females smoking 35 cigarettes or more daily were more likely to have had five drinks or more (13.2 percent) than females smoking 15–24 cigarettes daily (8.7 percent) and females smoking less than 15 cigarettes daily (6.7 percent). As was found with the "heavier drinker" classification discussed above, drinking five alcoholic beverages or more was considerably more common among males than among females, regardless of smoking status or amount smoked.

Summary

Overall, these results suggest that smoking is related to other unhealthy behaviors. Compared with nonsmokers, smokers are more likely to get little sleep, skip breakfast, not exercise actively, and drink heavily. In contrast, smokers were less likely to be overweight and less likely to snack daily than were nonsmokers, the more favorable weight status and snacking behavior tending to be most characteristic of lighter smokers.

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Technical notes

Data presented in this report were obtained from household interviews of the National Health Interview Survey (NHIS). These interviews were conducted among a probability sample of the civilian noninstitutionalized population of the United States. During 1985, 91,531 persons living in 34,844 households were interviewed. The total noninterview rate for the NHIS was about 4.3 percent, including 2.6 percent due to respondent refusal and the remainder due to failure to find an eligible respondent at home after repeated calls. Detailed information on the design of the survey is available elsewhere (NCHS, 1986, 1988).

Questions concerning health promotion and disease prevention were asked of a subsample, one person aged 18 years and over per family. This report is based on data for the 32,517 subsample respondents who were 20 years of age and over. Self-reporting was generally required for these questions, but proxy responses were accepted when subsample persons were physically or mentally incapable of answering the questions for themselves. Persons with unknown health practice characteristics are excluded only from the analysis for that health practice. The response rate for the NHIS Health Promotion and Disease Prevention Questionnaire was about 90 percent. Item nonresponse for the data discussed in this report ranged from 0.5 percent for breakfast to 3.6 percent for desirable weight.

The measure of desirable weight used in this report was based on the 1983 Metropolitan Life Insurance Company (MLIC) standards of desirable weights for men and women, which were derived from the 1979 Body Build and Blood Pressure Study conducted by the Society of Actuaries (MLIC, 1983). Desirable weights are shown for small, medium, and large frames for men and women. For NHIS analyses, the midpoint of the desirable weight range for the medium frame category was used as the "desirable weight" for a particular height, with certain adjustments. The MLIC standards were developed based on weight in indoor clothing and height with 1-inch heels. Respondents to the NHIS were asked to report their height without shoes and their weight without clothes or shoes. To compensate for these differences, the MLIC standards were adjusted by subtracting 2 pounds from the midpoint of the medium frame category for both sexes and subtracting 1 inch from the height.

Because the estimates shown in this report are based on a sample of the population rather than on the entire population, they are subject to sampling error. Standard errors appropriate for estimated percents of persons are shown in table I. Population sizes for each of the smoking groups are shown in table II.

To understand better the limitations of the estimates pre-

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Table I. Standard errors, expressed in percentage points, of estimated percents: 1985 National Health Interview Survey of Health Promotion and Disease Prevention

Base of percent in thousands	Estimated percent				
	2 or 98	5 or 95	10 or 90	20 or 80	50
50	5.1	8.0	11.0	14.7	18.4
70	4.3	6.8	9.3	12.4	15.5
100	3.6	5.7	7.8	10.4	13.0
300	2.1	3.3	4.5	6.0	7.5
500	1.6	2.5	3.5	4.6	5.8
700	1.4	2.1	2.9	3.9	4.9
1,000	1.2	1.8	2.5	3.3	4.1
5,000	0.5	0.8	1.1	1.5	1.8
10,000	0.4	0.6	0.8	1.0	1.3
20,000	0.3	0.4	0.6	0.7	0.9
30,000	0.2	0.3	0.5	0.6	0.8
50,000	0.2	0.3	0.3	0.5	0.6
100,000	0.1	0.2	0.2	0.3	0.4

sented in this report, data users are encouraged to familiarize themselves with the survey design, the methods used in estimation, and the general qualifications of the data, which are described in appendix I of Health Promotion and Disease Prevention, 1985 (NCHS, 1988). The NHIS Health Promotion and Disease Prevention questionnaire is shown in appendix III of that report.

NOTE: A list of references follows the text.

Table II. Number of persons 20 years of age and over by smoking status, number of cigarettes smoked daily, and sex: United States, 1985

Smoking status, number of cigarettes smoked daily, and sex	Number in thousands
Both sexes, all smoking statuses[1]	163,693
Never	71,590
Former	39,918
Current	48,792
Less than 15 cigarettes	14,982
15–24 cigarettes	20,267
25–34 cigarettes	6,597
35 cigarettes or more	6,524
Male, all smoking statuses[1]	77,187
Never	26,293
Former	24,086
Current	25,021
Less than 15 cigarettes	6,421
15–24 cigarettes	10,193
25–34 cigarettes	3,881
35 cigarettes or more	4,298
Female, all smoking statuses[1]	86,505
Never	45,297
Former	15,831
Current	23,771
Less than 15 cigarettes	8,561
15–24 cigarettes	10,074
25–34 cigarettes	2,716
35 cigarettes or more	2,227

[1] Includes unknown smoking status.

Suggested citation

National Center for Health Statistics, C. A. Schoenborn and V. Benson. 1988. Relationships between smoking and other unhealthy habits: United States, 1985. *Advance Data From Vital and Health Statistics.* No. 154. DHHS Pub. No. (PHS) 88–1250. Public Health Service. Hyattsville, Md.

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Centers for Disease Control
National Center for Health Statistics
3700 East-West Highway
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PRIVILEGED AND CONFIDENTIAL
ATTORNEY WORK PRODUCT

PROPOSALS TO SUBJECT CIGARETTES TO FDA OR CPSC JURISDICTION

Tobacco products are generally exempt from regulation by the Food and Drug Administration (FDA) and Consumer Product Safety Commission (CPSC). Anti-tobacco advocates apparently have chosen FDA regulation of cigarettes as one of their major policy objectives, and they are currently engaged in two efforts to subject cigarettes to FDA jurisdiction. First, petitions have been filed with the FDA urging it to assert jurisdiction over low tar/low nicotine cigarettes and the proposed RJR smokeless device. Second, Congressman Bob Whittaker has proposed legislation -- H.R. 3294 -- to create a new FDA regulatory regime for cigarettes.

A. Summary of Effects of Proposals for FDA Control

FDA's statute creates specific and distinct regulatory schemes for classes of products: foods, drugs, cosmetics, medical devices, veterinary medicines, and so on. The scheme to be applied to a product follows from the category to which that product is assigned.[1] If cigarettes are to be brought within

[1] In other words, FDA cannot create a new scheme for a new type of product, but may only apply an established
[Footnote continued on next page]

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traditional FDA regulation, they must be determined to be either "drugs" or "foods," and regulated as such. Under either classification, FDA could be compelled to take various actions which could well be tantamount to immediate and total prohibition of cigarettes:

1. If viewed as "drugs," cigarettes would probably be ruled to be "new drugs" for which FDA approval of a application is required by law. To obtain such approval, the application would need to contain extensive animal toxicology, as well as human clinical data, sufficient to demonstrate that the products are safe and effective for their intended use. Until such approval was obtained, the cigarettes would be illegal and marketing could be prohibited by FDA.

2. If viewed as "foods" or "drugs," cigarettes could only contain ingredients that are both safe and suitable for their use. FDA would have authority to rule on whether sufficient data exists to establish that the various additive ingredients used in the products are safe and appropriate. Again, until FDA has approved the various ingredients, their use could be prohibited and any cigarette product containing them would be subject to legal action.

[Footnote continued from previous page]
scheme that Congress designed as generally appropriate for a class of products.

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3. If viewed as "drugs," cigarettes would have to be labeled with adequate directions for use and adequate warnings against unsafe use, as determined by FDA. If FDA concludes that such labeling cannot be written for understanding by law persons, it may classify cigarettes as a "prescription drug," thereby prohibiting sale except on a physician's specific order for an individual patent.

In addition to these problems, subjecting cigarettes to the regulatory framework designed by Congress for drugs or foods would impose other significant burdens:

1. Whether cigarettes are classified as "foods" or drugs," manufacturing plants for such products would be subject to FDA's "Good Manufacturing Practices" regulations and inspection at any time by FDA inspectors. If cigarettes are viewed as "drugs," these manufacturing sites would also be required to register as drug establishments

2. If classed as "drugs," companies would be subject to extensive reporting regulations under which adverse reactions from using such cigarettes would be required to be publicly filed with on a timely basis.

3. If cigarettes were viewed as "drugs," all changes in product formula or manufacturing procedures might also be subject to preclearance by FDA.

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B. The Whittaker Bill

H.R. 3294 creates a new regulatory structure for tobacco products. Under this legislation, FDA would have the power to: (1) limit or ban any additive that is "unsafe and presents unnecessary increased risks to health" (and tobacco itself could be found to be an additive); (2) prohibit "any added poisonous or deleterious substance which may render [a tobacco product] injurious to health; (3) establish limits on the levels of tar, nicotine, carbon monoxide, and "other harmful constituents," and require disclosure of the amounts of those constituents on packages "or by other means"; (4) mandate disclosure of additives, require a package warning regarding addiction and take action against false and misleading labeling (and possibly advertising); and (5) tightly regulate tobacco manufacturing processes to prevent contamination of tobacco products. The Whittaker bill would also establish a national minimum age of 18 for purchase of cigarettes, ban sampling and coupons, and allow the federal government to regulate the "form, manner and sale of tobacco products."

C. History of Exclusion of Cigarettes from FDA and CPSC Jurisdiction

Congress has correctly recognized that the balance to be struck between the alleged health risks of

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smoking and the unquantifiable pleasure that people derive from smoking is a political issue that should not be delegated to the technical expertise of any federal agency. As a result, Congress has struck this balance itself by permitting the continued sale of cigarettes while insisting on increasingly detailed warnings. The fact that virtually every American is well aware of the alleged risk of smoking -- both from the mandated warnings on the packages and advertising, as well as the public debate over smoking and health -- demonstrates that there is no reason to second-guess that decision and call for some special regulation by FDA or CPSC.

Moreover, the laws that those agencies enforce were written by Congress for entirely different types of products. Cigarettes do not fit within the schemes established for foods, drugs, or durable consumer products. It makes no more sense to subject cigarettes to FDA's jurisdiction by calling them "foods" or "drugs" than to call cigarettes "securities" and so vest responsibility in the SEC.

1. FDA

FDA has recognized that the regulation of cigarettes is not within its legal authority. In December 1977, FDA rejected a petition by Action on Smoking and Health ("ASH"), calling for the general regulation of cigarettes by FDA as drugs. This action

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was subsequently upheld by Action On Smoking Health v. Harris, 655 F.2d 236 (D.C. Cir. 1980).

FDA ruled that Congress intended products to be regulated as "drugs" only when those who manufactured or sold them made specific claims of therapeutic benefit or an intent to affect the structure or function of the human body. Cigarettes, for which no such claims have been made, did not meet this statutory standard for regulation as a drug.

FDA similarly rejected a subsequent petition by ASH calling for the regulation of filter cigarettes as "medical devices". Again, FDA looked to see whether these products met the broad legal standard that Congress adopted to define FDA's jurisdiction. In that case, FDA held that the filter was not a device unless the manufacturer made some specific therapeutic claims for it. The claim that a filter simply reduced tar and nicotine in the cigarette smoke was not considered by FDA to be a therapeutic claim for purposes of the FDA act. (The issue of filter cigarettes is once again before the FDA in connection with a new petition by the Coalition on Smoking and Health.)

This is not to say that FDA has been reluctant to regulate a particular cigarette where a manufacturer has attempted to sell his product based on some specific health claim. For example, in the 1950s, FDA acted

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against cigarettes which made claims that they would "prevent respiratory diseases, common colds," or would curb the appetite and lead to weight reduction. The critical point is that FDA recognizes its statute establishes regulatory standards and requirements designed for classes of products, and that the scheme applicable to one class may make no sense when applied to another class of product. Thus, drugs must be shown efficacious for their intended therapeutic purpose, while foods do not because they have no such therapeutic purpose. Cigarettes as traditionally marketed simply do not fit within any of the regulatory schemes designed by Congress and assigned to FDA.

FDA has clearly been correct in assessing the limits of its jurisdiction, and Congress should not now try to force cigarette regulation into a system of FDA regulation that is not the appropriate vehicle for balancing the alleged risks and clear pleasures of smoking. First, it is clear that cigarettes are not a "food" where FDA regulation is primarily concerned with the issue of adulteration -- unintended contaminants in a product. Although the cigarette industry has been criticized on many points, no one has suggested that there is any such adulteration problem in our manufacturing practices. In contrast to the horror stories that have circulated for many years -- and are

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still common -- in such industries as meatpacking, there is no evidence that there is any need for government action in maintaining the cleanliness in cigarette manufacturing plants. (In this connection we might invite the Congressmen to come to Richmond to visit our facility.) In short, if the issue is one of protecting the American consumer from "adulterated" products, the government should spend its time and money in other industries where the problems of potential adulteration are much greater and where the products are used by consumers (including children) without any warning that they may contain such a health hazard.

Similarly, it is clear that it would be inappropriate to regulate cigarettes as "drugs". FDA's system of regulation has been created to consider the safety and efficacy of "drugs" which are intended for therapeutic purposes. As noted above, that distinction has been followed by FDA in regulating only those cigarettes which make claims that they actually treat or prevent some disease. But that issue is very different from the naked attempt to regulate cigarettes which make no such therapeutic claims. Such regulation would be totally inappropriate because FDA has no ability to make a "risk/benefit" determination in the case of cigarettes. Unlike "drugs" which are intended for a specific therapeutic purpose -- where the risks of

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unintended side effects can be balanced against the efficacy of the drug in providing its therapeutic effects, cigarettes are simply recreational products which give people pleasure.

In this regard, it is crucial to note that the fact that cigarettes contain nicotine and nicotine has some pharmacological action obviously does not classify the product as a "drug" as that term is used in FDA regulation. Many products contain chemical compounds which have such a pharmacological action. For example, coffee contains caffeine which, in sufficient doses, can be a powerful stimulant. And FDA does regulate pure caffeine when it is sold as a "drug" -- that is, when a company manufacturers a caffeine product such as "No-Doz" and sells the product to keep consumers awake. But no one at FDA or Congress has ever suggested that FDA regulate coffee as a drug.

D. CPSC

For many of the same reasons, the regulation of cigarettes should not be entrusted to the CPSC. Congress made this decision when it enacted the CPSC Act but expressly excluded tobacco. Despite the suggestions of our critics that this was simply a naked political compromise, the fact is that CPSC was never intended to have any special expertise that would lend itself to the regulation of cigarettes.

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The principal role of the CPSC is to require corrections, or to warn about, hidden defects in the design or function of some product. The CPSC is not intended to be an organization which bans products which have some risk. Most notably, CPSC has not acted to ban items used for simple pleasure and recreation except where the manufacturer could take some action to correct a design defect or to warn unaware consumers about the risks of a product.

Thus, for example, CPSC may well require the manufacturers of football helmets to correct some hidden defect in the design which the manufacturer can change without impinging on the practical use of the helmet. In other cases, CPSC may require the manufacturer to provide a warning of some unknown danger in the improper use of the helmet. But CPSC has never attempted -- and has no special competence -- to try to ban footballs themselves even though everyone knows that there are certain demonstrable risks in the game and that each year children are permanently injured as a result. Like Congress, CPSC has correctly recognized that it has no ability to make the basic judgment that a particular activity, or product, should be banned outright simply because there are risks which cannot be properly weighed against the unquantifiable pleasure of a particular activity.

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E. Ingredients

As a fallback position, our opponents may attempt to secure FDA regulation over the non-tobacco ingredients added to cigarettes. Such legislation, unless it contained "grandfather" provisions exempting existing ingredients, could obviously prove troublesome. Our response should be that, as described in greater detail in the memorandum concerning ingredients, the ingredients added to tobacco are, for the most part, "GENERALLY RECOGNIZED AS SAFE" (GRAS)(although this is in a food rather than inhalation situation); that there is no scientific evidence that the minute amounts of ingredients in cigarettes add, in any way, to the alleged risks of smoking; and that, perhaps most importantly, Congress decided only three years ago that this matter should be handled by having HHS review the list of all ingredients and then making recommendations. The companies provided HHS with that information, but to date HHS has not indicated that there are any problems with the ingredients list.

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DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

HF-43

Food and Drug Administration
Rockville MD 20857

September 29, 1987

The Honorable Henry A. Waxman
Chairman, Subcommittee of Health
and the Environment
Committee on Energy and Commerce
House of Representatives
Washington, D.C. 20515

Dear Mr. Waxman:

This is in further response to your letter of July 1 concerning two nicotine-containing products, "IPCO Creamy Snuff" and "Masterpiece Tobacs." I would first like to respond to your inquiry concerning the extent and limits of the Agency's general authority over food and drug products containing nicotine. The Agency may assert jurisdiction over nicotine-containing food or drug products if those products meet the relevant definitions of the Federal Food, Drug, and Cosmetic Act (FD&C Act). A more complete discussion follows.

Under section 201(g)(1) of the FD&C Act, 21 U.S.C. 321(g)(1), "drugs" are defined as: (A) articles recognized in an official compendium, (B) articles intended for use in the diagnosis, cure, mitigation, treatment or prevention of disease, (C) articles (other than food) intended to affect the structure or a function of the body, and (D) articles intended for use as a component of a drug. Under 201(f) of the FD&C Act, 21 U.S.C.321(f), "food" is defined as: (1) articles used for food or drink, (2) chewing gum, and (3) articles used for components of food.

The circumstances in which nicotine-containing products have satisfied these definitions are limited. Historically, FDA has not considered traditional tobacco-containing products, as customarily marketed, to be subject to regulation under the FD&C Act. Examples of these types of products are smoking tobacco, cigarettes, cigars, chewing tobacco, and snuff. On December 5, 1977, FDA denied a citizen petition that requested that it assert jurisdiction over traditionally-marketed forms of cigarettes as drugs. This denial was upheld in Action on Smoking and Health v. Harris, 655 F.2d 236 (D.C. Cir 1980).

FDA has in the past, however, regulated tobacco products as drugs when health claims were made by the manufacturers or vendors, such as representations that tobacco products were effective for the prevention or treatment of respiratory or other diseases or for weight reduction. For example, FDA brought an action against cigarettes that contained tartaric acid, a component that was represented to be effective for combatting obesity. The court held that these cigarettes were indeed a drug because of weight-reducing claims made in advertising and on the cigarette packaging. (See United States v. 354 Bulk Cartons Trim Reducing-Aid Cigarettes 178 F. Supp. 847 (D.N.J. 1959).

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Another example of FDA regulatory action regarding nicotine-containing products is that on February 9, 1987, FDA sent a regulatory letter to the manufacturer of "Favor Smokeless Cigarettes" and "Favor Smoke-Free Cigarettes," which contained nicotine purportedly derived from tobacco, but no tobacco. These products were represented as a novel nicotine delivery system and a method of administering nicotine by inhalation of nicotine vapor. In these products, a fibrous plug impregnated with a nicotine solution was inserted within a small plastic tube resembling a conventional cigarette in appearance. The regulatory letter advised the firm that "Favor" is a nicotine delivery system intended to satisfy a nicotine dependence and to affect the structure and one or more functions of the body, and that because of its intended uses, "Favor" is a drug. FDA also advised that, in fact, "Favor" is a "new drug" within the meaning of section 201(p) of the FD&C Act because its composition is not generally recognized, among qualified experts, as safe and effective for use under the prescribed or recommended conditions as described in its labeling. The manufacturer had disagreed with our conclusion that "Favor" is a new drug but did suspend distribution and promised to inform the Agency in advance if it contemplated resumption of marketing.

As you know, FDA has also regulated "Nicorette" as a drug. It is clearly intended and labeled as a smoking deterrent to satisfy a nicotine dependence, following the cessation of smoking. "Nicorette" was originally developed and marketed in Europe, and in the early 1970's, clinical investigators in this country became interested in studying the product. They submitted to FDA investigational new drug applications in an effort to determine its safety and efficacy as a smoking deterrent. Soon after, Merrell Dow Pharmaceuticals, Inc., became interested in marketing "Nicorette" as well and submitted safety and efficacy data to the Agency. The NDA for "Nicorette" was approved by FDA on January 13, 1984.

Masterpiece Tobacs primarily consists of gum, tobacco, sweeteners and flavoring. It looks, tastes, and chews like chewing gum and because of the flavors and sweeteners in the gum, the saliva is likely to be swallowed as in gum chewing rather than expectorated. Further, it is unlike traditional smokeless tobacco products. Thus, we believe that Masterpiece Tobacs is a chewing gum and a food under the FD&C Act.

We also believe that this product is an adulterated food because it contains the ingredient tobacco, which is not generally recognized as safe (GRAS) for use in foods, and there is no regulation authorizing the use of tobacco in chewing gum. This product is, therefore, subject to legal action because it contains a food additive deemed unsafe within the meaning of section 409 of the FD&C Act and is, thus, considered to be adulterated within the meaning of section 402(a)(2)(C) of the FD&C Act. I have enclosed a copy of a letter to Mr. Stuart Pape that addresses the product Masterpiece Tabacs.

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We are currently pursuing an investigation of IPCO Creamy Snuff to further evaluate its status with respect to the Federal Food, Drug and Cosmetic Act. In this regard, an assignment was sent to our Philadelphia District Office on July 31, 1987. Preliminary information indicates that the product may not be subject to the drug sections of the Act, but we are continuing our investigation.

I hope the preceding information has been helpful. If I can provide you with additional information, please don't hesitate to call me.

Sincerely yours,

Frank E. Young, M.D., Ph.D.
Commissioner of Food and Drugs

Enclosure

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DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

HF-43

Food and Drug Administration
Rockville MD 20857

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Sincerely yours,

Frank E. Young, M.D., Ph.D.
Commissioner of Food and Drugs

Enclosure

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RURAL DEVELOPMENT, AGRICULTURE, AND RELATED AGENCIES APPROPRIATIONS FOR 1989

HEARINGS

BEFORE A

SUBCOMMITTEE OF THE

COMMITTEE ON APPROPRIATIONS

HOUSE OF REPRESENTATIVES

ONE HUNDREDTH CONGRESS

SECOND SESSION

SUBCOMMITTEE ON RURAL DEVELOPMENT, AGRICULTURE, AND RELATED AGENCIES

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JOE SKEEN, New Mexico
VIN WEBER, Minnesota

ROBERT B. FOSTER and TIMOTHY K. SANDERS, *Staff Assistants*

PART 8

AGRICULTURAL PROGRAMS

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Mr. WATKINS. Thank you, Dr. Young.
Thank you, Mr. Chairman.
Mr. WHITTEN. At this time, we will recess for the vote in the House and come back.
[Recess taken.]
Mr. WHITTEN. The subcommittee will come to order.
Mr. Durbin?
Mr. DURBIN. Thank you, Mr. Chairman.
Dr. Young, thank you for joining us again this year. I have enjoyed working with your agency. You have extraordinary responsibility, and I feel that you and the people who work with you do an extraordinarily good job which is perhaps unheralded. I hope that this hearing today and the reporting of it will help to bring the recognition which you deserve for a job well done.
Dr. YOUNG. Thank you.

TOBACCO

Mr. DURBIN. Your opening remarks, particularly the first page, describes what you have called a "particularly broad mandate" which requires that you educate consumers about the safety of products, and regulate them so that the consumers can participate in protecting their own health.
As an overview of your agency, it would appear that you generally have three options at your disposal: to prohibit certain products, to regulate others, and perhaps in conjunction with either or both of those responsibilities to at least require disclosure so that consumers can make an educated choice.
We have heard during the course of the questions asked of you by my colleagues here a description of many different activities and products, and the role of your agency has been plumbed and explored in terms of regulating those products. I am going to ask you some questions about one particular product that concerns me. It is a product which is consumed by millions of Americans, but its contents are not disclosed. In fact, the contents are almost totally secret, and the product itself is exempt from virtually all health regulation.
We provide a government subsidy for this product, and we know that it is at least contributory or perhaps causal in the deaths of 350,000 Americans a year. It would seem that if you looked at the spectrum of products sold in America, it stands out as an extraordinary exception to your mandate under the FDA and our concern as a government.
The product, of course, is tobacco and tobacco products. There have been tests over the years in court as to whether or not tobacco is included in the Food, Drug and Cosmetics Act, and the courts have generally concluded it is not. In fact, I think they have concluded unanimously that it is not an ingested food, nor is it a drug taken for an illness or to change some type of body structure.
I would like to ask you from a different perspective whether you can give me any medical or scientific basis for this exception that tobacco not be included as a product regulated by your agency.
Dr. YOUNG. I will ask Tom Scarlett to comment from a legal standpoint, but I suspect that from what you have said, that is not

the direction you are
aspect?
Mr. DURBIN. I think
tobacco is not include
asking from a medica
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Dr. YOUNG. Dr. Koo
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Dr. YOUNG. Yes.

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the direction you are coming from. Is that correct, not the legal aspect?

Mr. DURBIN. I think the legal interpretation is fairly clear that tobacco is not included in your legislative mandate. But I am asking from a medical or scientific viewpoint with your public health concern whether or not you can see any basis for this exception.

Dr. YOUNG. Dr. Koop and all of the Surgeon Generals that preceded him in recent years have focused on cigarette smoking as one of the major areas for concern. He and others before him have advocated a smoke free environment by the year 2000.

In my own experience as a pathologist, I have had the opportunity of dealing with and autopsying a number of people with lung cancer. There is no question that there have been certain diseases medically that have been implicated, whether directly or indirectly, with the consumption of tobacco. I don't think anyone will dispute that as a consensus. I think some will dispute that in different quarters. There is always dissent one way or another on the cutting edge of science.

The question comes in then how to focus on areas that do not seem to be within a mandate of a particular law. The chairman and I were talking about that earlier in regard to defining what a level of risk is when we were talking about the de minimis policy, how you handle that.

I had to deal with that—parenthetically, but it is directly related—when we were looking at a compound that I first dealt with when I came into the agency, acrylonitrile. Mr. Scarlett said this is under the food safety provision, not the Delaney Clause. It can be approved. There is a one in a million upward bound risk of an additional cancer.

A few months later, I looked at some of the colors. The upward bound risk was one in 19 billion, and they said that should be banned. I was thunderstruck. How could you ban one with a low risk and approve one with a little bit higher one.

I began to learn then that the law puts things in different packages. I said in attacking the de minimis approach that as a physician, it gives me no comfort to find someone dying under one provision appropriately or dying the same way under a different provision inappropriately. If you are dead, you are dead.

That comes back to the heart of your question of how do you deal with a very complicated issue.

From a medical standpoint, we have taken the substitution in the drug that we approved, Nicorette, as appropriate because there you could determine how this could substitute, and then see a person weaned if that person wanted to. However, my concern would be that it doesn't look like it is possible to regulate it under the Food, Drug and Cosmetic Act even though smoking, I think, has been widely recognized as being harmful to human health.

Mr. DURBIN. I would agree with the conclusion that it is not covered by the act. I would say that I have difficulty with that, though. I think the act was carefully crafted so that we could still have questions from my colleague from Hawaii about your investigation of alcohol and the additives to alcoholic beverages.

Dr. YOUNG. Yes.

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Mr. DURBIN. We know that that is used almost primarily for recreational uses and the like. We enjoy alcoholic beverages in this country. It is not a food per se, a nutrition source, although I am sure there is some nutrient value to a glass of beer and perhaps other alcoholic beverages.

However, what I am driving at is this. It strikes me that if on the one hand the drug, nicorette, or the drug, nicotine, is regulated by FDA, we know for a fact that nicotine is contained in virtually every tobacco product. We also know that because of your analysis of this nicorette or nicotine, you came to the conclusion that there should be some regulations perhaps as it relates to the sale or prescription of that drug.

Dr. YOUNG. Yes. That was really used to treat a nicotine withdrawal.

Mr. DURBIN. So, as you look at the tobacco product just on this aspect—and there is one other aspect I would like to address—we are dealing with a drug that is administered in an extraordinary way with little or no regulation. There are no prohibitions—I am sure there are some on the local and State basis, but none on a Federal basis that I am aware of—on the sale of tobacco products.

In this situation, virtually anyone of any age could come into possession of this drug and ingest it, but having done it by cigarette rather than by drug form, nicorette, they totally escape any Federal regulation. There seems to be some basic inconsistency here.

If our public goal is the public health of America, then the form of the drug as it is administered should be secondary to the goal, as I understand it. Perhaps I am taking you further than you are prepared to go today.

Dr. YOUNG. Tom, could you comment on that? In part, it is a legal question as to whether it is included under the act. Could you give us some history on that?

Mr. SCARLETT. Well, I don't want to belabor a point that Mr. Durbin has already acknowledged, namely, that the act draws lines and we have to respect them. I guess the one reaction that I was having to what you were saying was that what is fairly important in FDA law is whether a product has a therapeutic purpose, at least if you are talking about something that might be a drug.

Nicorette is intended to, in effect, treat cigarette addiction. Cigarettes themselves are not used for a therapeutic purpose as that concept is ordinarily understood. The opposite argument has been made and, I am sure, will be made over the years.

The other reaction I was having was that based on an experience that the CPSC had when it once undertook to ban bullets, a likely reaction that Congress might have if FDA attempted to regulate cigarettes would be to amend the FDA's law so that it could not.

Mr. DURBIN. I would agree that if you embarked on that course, you would definitely want some legislative support and, at this moment, it is not forthcoming. But I am attempting to explore that subject to determine whether or not it should be.

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The other area beyond the addictive quality of nicotine and tobacco is one that may be even more pernicious, and that is the question of tobacco additives. As I understand it, there is virtually no disclosure, certainly not to FDA, of additives in tobacco, whether they be pesticides, poisons, carcinogenic, or the like. So, in fact, there may be a product being sold on the market in America which could pose great health risk.

There are now cases pending involving liability of tobacco companies. I will not address any one specifically, because I think that would be uncalled for. But there have been disclosures in those trials that, in fact, sometimes there have been extraordinarily high pesticide residues on tobacco that has been sold to the American public.

You and I have talked for over a year now on pesticide levels on tomatoes coming in from Mexico and the efforts that we have made and the millions of dollars spent to protect the American consumers from any bad health effects. I think these efforts are well documented. Yet, in this instance, a chemical additive is going virtually unnoticed with no requirement for disclosure.

So, beyond the drug aspect of nicotine itself, what would you say to the concern over these chemical additives that are in tobacco products?

Dr. YOUNG. As far as I know, the regulation would be under the Bureau of Alcohol, Tobacco, and Firearms in regard to anything that would be there. To the best of my knowledge, one is not required to disclose any of the additives.

I think it is fair to say that regardless—I am now talking of the harmful chemical additive—if it were unchanged in consumption, it would be according to the principles that we have used in cancer evaluation—I will just stick with that field—that if it were not destroyed or modified by the smoke and heat, once you look at one route of entry, whether it be respiratory, skin, or GI, we have to assume that the residue is similar by any route.

I would be happy to go to my colleagues in BATF and see what I can learn about the ingredients. It is not an area that I am familiar with.

Mr. DURBIN. I know you don't need to broaden your already broad mandate, but if you would make some inquiry there, I would appreciate it.

Dr. YOUNG. I would be happy to, and I will provide that for you.

[The information follows:]

REGULATION OF ADDITIVES IN CIGARETTES

FDA staff have contacted the Bureau of Alcohol, Tobacco and Firearms (BATF) and has confirmed that BATF does not test cigarettes for pesticides or other chemical contaminants. It should be noted that The Federal Trade Commission has specific responsibilities regarding the labeling and advertising of tobacco products, but as far as we know they are not concerned with additives in cigarettes.

Mr. DURBIN. One last question on this particular subject. The chairman referred earlier to the Delaney Clause. We are familiar with its requirement, that the FDA remove any additives which could induce cancer in man or animal. We also know that unfortu-

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nately, we have about 80,000 lung cancers a year in the United States from smoking.

At the present moment, the way the Delaney Clause is constructed as an adjunct to the Food, Drug, and Cosmetic Act, you are powerless to address the carcinogenic aspects of smoking or tobacco.

Dr. YOUNG. That is correct. That would be viewed as being outside the scope of the Food, Drug, and Cosmetic Act as it is constructed today, and it would be a lacuna in regard to public health. I think that is why you see Surgeon General Koop and other Surgeon Generals going out and focusing at it with health education. That seems to be the way that is available to the public health service at this time.

Mr. DURBIN. Well, it is certainly beyond de minimis. When you think of the efforts that were made with a product like saccharine, for example, to alert the public of its potential carcinogenic qualities when consumed in quantity, it shows that we have really left a gap when it comes to public health as it relates to tobacco and smoking.

Dr. YOUNG. Yes. There is a warning, as you know, on the package, but that is the limit at this point.

Mr. DURBIN. Yes, and the warning is being used as a defense in many of these law suits on the basis that the smoker is assuming the risk. There is another aspect, of course, to selling an addictive product where perhaps a conscious and free decision is not being made each time as to the consumption of the product. But the courts will have to decide that.

I thank you for your comments, I am sure we are going to explore this a little further perhaps in the authorizing committee.

Dr. YOUNG. It is a tough issue, a very difficult issue.

PESTICIDES ON IMPORTED FOODS

Mr. DURBIN. Let me switch to an area that may be more familiar turf for us. I am very happy with the response which your agency has given me on the inspection for pesticides on imported food products. For those who are not familiar with it, several members of the staff joined me in a tour of several of your facilities. I was impressed with the people, the equipment, and the attitude of your employees. I think they do an extraordinary job.

Dr. YOUNG. They do an excellent job.

Mr. DURBIN. They do.

Dr. YOUNG. We are blessed with having outstanding people in the rank and file.

Mr. DURBIN. And they are behind the scenes, and I don't think most people appreciate that.

I had suggested that you might consider a procedural change when it came to the inspection of imported food products.

Presently under surveillance—and you can correct me if some of these facts have eluded me after a few months—you are taking samples of imported fruits and vegetables. After those samples are analyzed, if you find anything that is untoward or suspicious, you can put a producer on what is known as compliance status whereby they will be regularly inspected as they send in these products.

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INGREDIENTS AND PESTICIDES

During the past year, the plaintiffs in the liability cases have begun to make claims based on the fact that the companies add ingredients other than tobacco to the cigarettes and that cigarettes contain minute residues of the pesticides used in the agricultural, and (some contend) manufacturing, processes used to grow and store tobacco. (These issues were not tried in Cipollone because we were able to convince the Court that Edell's last minute effort to do so was too late. By contrast, the issue of ingredients and pesticide residues was central to the Horton case in Mississippi against American which resulted in a hung jury.)

At the same time, there have been a number of proopsals in Congress that regulation of ingredients and pesticide residues by some agency of the federal government would be one way to curtail the industry. For example, the Whittaker bill (H.R. 3294) would give the Secretary of Health and Human Services the authority to ban any additive which is "unsafe and presents unnecessary increased risks to health" as determined by the Secretary with "the advice of experts qualified by

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scientific training and experience to evaluate the safety of tobacco additives."

As described below, these efforts of the tort plaintiffs and the Congress to fasten liability or additional regulation on the industry generally ignore the fact that the ingredients and pesticide residues in cigarette smoke are present in such minute amounts that they do not pose any health hazard.

A. INGREDIENTS

The Scientific Background

There is nothing new in the fact that cigarette manufacturers add various ingredients to their products. Ingredients have been added to tobacco to influence its flavor since the Indians first introduced smoking to the European explorers. The manufacturers use such ingredients for a variety of purposes:

-- Casings and humectants are added to replace sugars lost during the curing of the leaf, to help tobacco retain moisture, to aid in processing, and to make a smoother, milder smoke. These ingredients include various foods, or ingredients approved for food use, such as sugars, corn syrup, glycerol and propylene glycol.

-- Flavor ingredients are added to fortify natural tobacco flavors and to produce special flavors

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such as vanilla or menthol. Again, these flavor additives include natural food products or ingredients approved for use in food products.

-- Various processing aids are used to adjust nicotine content among different crops of tobacco (ammonia) or to expand tobacco (carbon dioxide). The residues of such processing aids are extremely small, however, because the very process in which they are used expends them long before the tobacco is made into cigarettes.

The use of such ingredients in cigarettes is generally premised on their long use in foods without adverse effects. This has been the approach not only of the tobacco industry in the United States, but also of regulatory agencies in other countries such as the Hunter Committee in England. While there are obvious differences between the use of additives in a product which is eaten and one which is smoked, the long history of the safe use of these products in food is evidence that the use of the same ingredients in cigarettes -- particularly in the minute amounts in which they are used -- does not constitute a serious health concern.

In response to the likely criticism that the industry has not biologically tested every ingredient used in cigarettes, we can emphasize that many of the 20 or so "major use" items which account for the vast

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majority of all ingredients added to cigarettes (about 90% by weight) -- humectants, sugars, sweeteners, and processing aids -- are included in the Kentucky reference cigarettes which have been used in inhalation and skin-painting studies. Thus, all of the biological testing conducted on such reference cigarettes in effect constitutes testing of the major use ingredients as well. None of that testing has demonstrated that the inhalation of such minute quantities of the additives -- with or without the accompanying tobacco smoke -- produces lung cancer.

The remaining flavor ingredients (special flavors, etc.) are used in such minute quantities that they are virtually undetectable in the content of cigarette smoke. To date, there has never been any evidence in any of the government's research or the "expert" opinion presented in any of the litigation that the addition of these minute amounts of such flavors changes the overall composition -- much less the carcinogenicity -- of the smoke.

The tobacco industry in general, and Philip Morris in particular, have acted responsibly with respect to the use of ingredients. Although we have not tested every ingredient in a separate smoke inhalation test, Philip Morris scientists have reviewed any literature on the possible toxicological effect of the

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ingredients and have tested the major use items for biological activity through Ames tests (which look for mutagenic changes in bacteria). Neither these tests, nor any test conducted by any outside party, have demonstrated that any of the ingredients Philip Morris currently uses constitutes a health hazard.

Finally, it should be noted that, over the years, the cigarette companies -- like food processers and others who use various ingredients -- have decided to remove certain ingredients from their products in response to possible health concerns. Coumarin -- an ingredient found in many foods and cosmetics -- is one such example. This is not to say, however, that Philip Morris or the other companies concede that the minute amounts of coumarin that had been used in cigarettes were harmful. Philip Morris was simply acting responsibly, along with the other companies, in eliminating coumarin from its products following reports that large amounts of coumarin might be toxic based on some animal experiments.

The Disclosure Issue

Although Philip Morris and the other cigarette companies closely guard their precise flavor formulas, the public has long been aware that cigarettes contain many ingredients in addition to tobacco. The use of

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such additives has been publicly discussed in patents which have been issued for various flavors, a 1953 report in Consumer Reports describing cigarette flavorings and casings, a 1959 article in Reader's Digest on cigarette manufacture, comments in the 1964 and subsequent Surgeon General Reports, and the comprehensive 1972 Leffingwell text, Tobacco Flavoring for Smoking Products. The last source -- a book written by a Reynolds' scientist with that company's assistance -- contained comprehensive listings of the flavorings used in tobacco products (although it, of course, did not disclose the formulas used in any specific Reynolds product).

The efforts of Philip Morris and other companies to protect their formulas from disclosure -- like the efforts of the flavor houses to protect the ingredients in their proprietary flavors -- is not part of any attempt to mislead or deceive the public. Rather, it merely reflects the importance of this information and the fact that there is no other legal protection for the particular combination of ingredients used in a particular product. In this respect, the cigarette industry is no different from any other industry which uses ingredients (e.g. the famous Coca-Cola formula).

Philip Morris has likewise complied with all efforts of the governments of a number of nations to

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review its use of ingredients. Specifically, Philip Morris follows the list of improved ingredients established by the Hunter Committee in England and the West German health authorities.

Similarly, since 1985 Congress has required Philip Morris and the other American cigarette companies to submit to HHS a combined list of all ingredients they use. That list has been updated annually since that first submission. While the combined list protects the precise list of ingredients used by any one company and the formulas for any one brand, it does advise HHS of the identity of all the ingredients used by all of the companies and thus would flag any potential danger.

Indeed, the cigarette ingredient list submitted to HHS is far more detailed a list of "ingredients" than the lists published by other manufacturers because the cigarette list breaks down the products added by the manufacturers into their respective constituents. It should thus be emphasized that while the HHS list totals more than 700 "ingredients", the tobacco companies do not add that many items as such. Rather, the industry adds a far smaller number of products (most notably, the proprietary flavors of various flavor houses) which in turn are made from many other constituents. As a result, many items on the HHS list are "used" in only

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trace amounts -- as little as one pound for the annual production of the entire industry.

For example, Philip Morris may use one pound of a certified food flavor made by an outside flavor house -- Vanilla No. 6 -- in processing ten thousand pounds of tobacco, just as a food manufacturer might use one pound of the same Vanilla No. 6 in producing a hundred pounds of cake mix. Philip Morris has determined, and in turn has advised HHS, that that one pound of Vanilla No. 6 is composed of twelve ounces of vanilla beans, three ounces of vanilla extract, one-half ounce of 3-Ethoxy-4-hydroxy-1-benzaldehyde (vanillin), and trace amounts of 10 other exotic sounding, but quite harmless, constituents.[1] Philip Morris lists each of those trace ingredients on its contribution to the combined industry list. But such detailed constituent information has never even been determined by the food manufacturer, much less conveyed to any government authority.

HHS, in turn, submitted the list of tobacco ingredients to the Office on Smoking and Health (OSH).

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[1] This, of course, is just a hypothetical. In any testimony we should emphasize that, even within the companies, the HHS list is only available on a need-to-know basis. More importantly, the list of ingredients used in any particular Philip Morris brand is available to only a handful of Philip Morris employees; and no one person has ready access to the complete formula in terms of both the identity of the ingredients and the quantities used.

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Although OSH has had the list for more than two years, they have not criticized any of the items on it. This is further evidence that there is no imminent health hazard from the use of ingredients which requires any additional government regulation.

B. PESTICIDES

It has long been known that there are pesticides residues in cigarette tobacco -- just as there are pesticide residues in virtually all of the food that we eat. Such residues necessarily result from the agricultural use of pesticides in the field. Because many of the pesticides are systemic -- that is, they are taken up through the roots of the plant into the leaves -- there is no way to eliminate such minute residues from tobacco or other food products. (Indeed, some pesticides are so stable that they transfer from one agricultural product to another: One can find residues of pesticides in milk and beef that result from the treatment of seeds to grow the corn that fed the cattle.) Pesticides are thus part of our daily diet (as well as the air we breathe).

At the same time, it is important to note that the use of these pesticides is closely regulated by the federal government. Since at least the late 1940s, the Department of Agriculture and then the Environmental

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Protection Agency have maintained an approved list of registered pesticides. In order to obtain such a registration, the pesticide manufacturer must demonstrate that its pesticide is safe and effective for use on the particular crop for which it is to be registered.

The government similarly promulgates regulations and provides practical advice as to how pesticides are to be used by a farmer. In order to participate in the government's price support program, tobacco farmers must certify that they have complied with these regulations both with respect to the type of pesticide used and the manner in which it was applied.

In addition to this program of government enforcement, the tobacco industry has, for 20 years, funded the efforts of North Carolina State University in monitoring pesticide residues both in warehouse inventories and finished cigarettes. The reports of N.C. State, which have been published annually, demonstrate that the amount of pesticide residues in tobacco -- like pesticide residues in the food supply in general -- have declined over the years.

There is no persuasive scientific evidence that the amount of pesticide residues in tobacco constitutes a health hazard. First, it is clear that the amount of pesticides in tobacco are not inordinately high. Even

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in the 1950s, when pesticide residues were far greater than they are today, an independent study cited by the Surgeon General reported that the amount of pesticides that would be inhaled by a smoker consuming 2½ packs of cigarettes a day was only about 1/10 the amount that he would inhale simply by breathing the air of New York City.

Finally, it should be noted that the only significant study to ascertain the possible effect of pesticide residues in cigarette tobacco concluded that they had no harmful effect. As part of the federal government's Tobacco Working Group research, cigarettes made from specially-grown pesticide-free tobacco were compared with Kentucky-reference cigarettes -- which had the normal amount of pesticide residues -- in a mouse skin-painting experiment. The research concluded that there was no difference in the biological activity of the condensate.

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FIRE-SAFE CIGARETTE ISSUE: AN OVERVIEW

The purpose of this overview is to set forth the basic positions of the industry with respect to various aspects of the fire-safe cigarette issue. It will endeavor to anticipate and provide responses to questions which may arise at a congressional hearing.

I. HISTORICAL BACKGROUND

This section will briefly summarize the recent history of this issue. In 1979 and 1980, contemporaneous with the filing of lawsuits involving cigarette-related fires, Andrew McGuire and others began to encourage legislation at the federal and state levels to require that cigarettes be made "fire safe". In support of this effort, many of the claims made by the proponents of the legislation were unfounded and were based upon misinformation. The proponents cited the presence of existing patents for fire-safe cigarettes and claimed that certain European and U.S. cigarettes (Sherman, More and Carlton) were "fire safe".

The industry's general response was to the effect that cigarette manufacturers strongly desire to reduce the incidence of accidental fires, including those involving the careless use of

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cigarettes and other smoking materials. The smoking public is aware of the potential fire risk presented by a lighted cigarette and 99.99% of cigarettes are properly extinguished. Nevertheless, through carelessness, accidents happen and tragic fires may result. The manufacturers had reviewed patent and scientific literature and based on their expertise did not believe an acceptable product could be made which would not start fires. The companies were independently pursuing research.

Through various activities, the Tobacco Institute endeavored to broaden the issue to focus on prevention efforts directed at all accidental fires. In addition, the Institute engaged in a cooperative research project with UFAC, a furniture manufacturers association, to investigate cigarette ignition of upholstered furniture materials.

The Institute developed contacts with fire services primarily at the state and local level. The focus of this effort was in two areas. First, direct support was given to local fire departments by providing public relations materials to assist the departments in fund raising and firemen recruitment, and providing audiovisual equipment, pamphlets, slides and films to aid in fire prevention presentations. The second area involved developing

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fire prevention materials targeted to those shown to be the most likely victims of home fires: urban low-income, elderly and disabled persons. Materials were also developed for use with elementary and high school students. The materials were designed to increase awareness of fire prevention and detection techniques, including promotion of the proper installation and maintenance of smoker detectors. The Institute's efforts were well-received by many in the fire services who have been generally supportive on a number of issues.

The industry continued to oppose legislative proposals in various states. The proposals called for "quick fix" legislation to what was obviously a very complex scientific matter. Most of the proposals would have required cigarettes to go out in a given time period and delegated the regulatory authority to a State Fire Marshall whose office had no expertise, limited facilities and inadequate resources. At the federal level, the industry met with Congressional and Senate proponents (principally Mass. Congressman Joe Moakley and Calif. Senator Alan Cranston in an effort to arrive at a sensible piece of legislation. The result was the Cigarette Safety Act of 1984 which created an Interagency Committee ("IAC") consisting of the Chairman of the Consumer Product Safety Commission, the U.S. Fire Administrator and the Assistant Secretary of

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Health and Human Services. A Technical Study Group ("TSG") was also created to perform an investigation "to determine the technical and commercial feasibility, economic impact, and other consequences of developing cigarettes and little cigars that will have a minimum propensity to ignite upholstered furniture or mattresses." Under the statute, the Technical Study Group was to report its findings to the IAC which in turn was to report to Congress. The 15-member TSG was comprised of representatives from the fire services, furniture manufacturers, the American Burn Association, the American Medical Association, DHHS, and 4 tobacco manufacturer representatives (Philip Morris, Lorillard, American and Reynolds). The TSG met periodically over a period of approximately 2 3/4 years (spending more than $2.1 million) and issued its final report to the IAC at the end of October 1987. In turn, the IAC filed its report and recommendations with Congress on December 23, 1987.

The TSG found that "it is technically feasible and may be commercially feasible to develop cigarettes that will have a significantly reduced propensity to ignite upholstered furniture or mattresses." (Emphasis added). It reached the conclusion that certain cigarette characteristics (smaller circumference, lower density tobacco, less porous paper, and reduced citrates in paper) were found in the laboratory to reduce ignition propensity. The

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Group also concluded that "the differences in ignition propensity among selected current commercial cigarettes are unimportant." The TSG recommended additional research, including (1) development of a standard test method to determine cigarette ignition propensity and (2) development of "knowledge" concerning "changes in the toxicity of smoke and resultant health effects from modified cigarettes." In making its report and recommendations to Congress, the IAC accepted the findings of the TSG and urged additional testing to determine commercial marketability of "less fire-prone cigarettes," plus an evaluation of any health risks smokers might incur from such cigarettes. Copies of the IAC report and the Executive Summary of the TSG report are available for reference.

The current status in Congress is that Mr. Moakley has introduced a follow-up bill into the House and there is a similar bill in the Senate sponsored principally by Senators Cranston (Ca.) and Heinz (Pa). On April 29, 1988, a bill supported by the tobacco companies was introduced by Congressman Boucher (Va.). A comparison of the pending legislative bills is addressed under a specific heading.

Bills have also been introduced in various states that generally call for creation of a performance test and give the

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companies a period of time to make products that pass the test. So far, no states have passed legislation and one was recently defeated in Minnesota.

The following sections will discuss areas of possible inquiry and provide response positions.

II. TSG RESEARCH, FINDINGS AND RECOMMENDATIONS

The findings, conclusions and recommendations of the TSG are set forth in the Executive Summary of the report. This section will capsulize the most relevant points and briefly discuss the research done and appropriate industry criticisms and comments.

The TSG conducted two important pieces of research using laboratory mockups of upholstered furniture materials. The work was performed principally by the National Bureau of Standards. In the first project, 12 commercial cigarettes were selected to include brands which had large market shares. This included one of the Marlboro brands. More and Carlton were also included because some researchers had claimed those brands were less ignition prone. The test results showed no statistically significant differences among the commercial brands. The TSG described the results by

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saying, "differences in ignition propensity among selected current commercial cigarettes are unimportant". Identification of the cigarette brands was kept confidential and not made known to members of the TSG or the public.

The second important project involved the laboratory testing of 41 experimental cigarettes which were specifically designed to determine if changes in cigarette construction would have an effect on ignition propensity. A description of the cigarettes and results of the laboratory tests are set forth in Tables 1 and 2 of the TSG's final report which are available for reference. Based upon the results of the test, the TSG found that reduced circumference, lower density tobacco (100% expanded tobacco), low porosity paper, and cigarette paper without the addition of citrates contributed to a reduction in ignition propensity. The experimental cigarettes which had the fewest ignitions were outside the commercial range of cigarette construction in terms of tobacco density, paper porosity and paper additives.

The TSG also performed mockup testing on a few patented cigarettes which did not ignite. The cigarettes were made by the patentees and were not examined to any extent by the TSG. The

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Group reported that, "Several patented approaches also offered directions for further investigation."

Based upon this research, the TSG made its finding, "that it is technically feasible and may be commercially feasible to develop cigarettes that will have a significantly reduced propensity to ignite upholstered furniture or mattresses." The TSG report went on to state that a valid and reliable test method is needed to measure reduced ignition in cigarettes. The report noted the wide lot-to-lot variation among upholstered furniture fabrics and cushion materials. This points up a major deficiency in the laboratory mockup testing, that is, there is considerable lack of uniformity between different lots of identical fabric and cushion materials made by the same manufacturer. Thus, questions can be raised regarding the validity of the test procedures used by the TSG and whether they have any relevance to furniture and mattresses found in the real world.

The report also acknowledged that consumer acceptability of the experimental cigarettes had not been tested. It noted that per puff tar, nicotine and carbon monoxide yields of some of the least ignition-prone experimental products were within the commercial range. However, the report stated that, "the toxicity

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of smoke from a future low ignition propensity cigarette needs to be addressed, as would the smoke from any substantially modified commercial cigarette, before its introduction into the marketplace."

The TSG report recommends additional research, including development of a standard test method to determine cigarette ignition propensity and to develop knowledge regarding changes in the toxicity of smoke and potential health effects from modified cigarettes.

The IAC made its recommendation to Congress for a two-year study to develop and test prototype cigarettes with reduced ignition propensity for consumer acceptability and smoke toxicity, and to develop a standard ignition propensity test method. The IAC also noted that a number of questions in these areas were left unanswered by the TSG final report.

A basic response to the TSG effort would be as follows:

-- Work by the TSG is helpful, but not finished.

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-- Definitive findings and conclusions should not be drawn on the basis of the research performed.

-- Important projects were not completed or attempted because of time and funding limitations.

-- The experimental cigarettes which showed reduced ignition propensity in the laboratory are markedly different from current commercial cigarettes.

-- Use of 100% expanded tobacco and very low porosity paper create major commercial feasibility difficulties:

- Puffing resistance is increased approximately 3 times the normal commercial range.

- Smoke composition can be expected to be altered.

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- Taste and aroma will be significantly affected.

-- As one moves away from experimental construction parameters towards less than 100% expanded tobacco and higher porosity paper, reduced ignition effects may be quickly eliminated.

-- There are serious questions concerning the validity of the test methodology used and its relationship to furniture and mattresses in actual use.

A November 10, 1987 letter from Dr. Charles to the CPSC discusses recommendations for needed research. This letter is available for review.

The TSG made a finding that it is technically feasible to manufacture cigarettes with reduced ignition propensity. This finding was based on the fact that the experimental cigarettes had been "manufactured" by the companies. The company representatives pointed out that the cigarettes had been made experimentally and not on high-speed equipment currently in use. They noted that

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technology may need to be developed in order to modify cigarette making equipment to incorporate drastic changes in cigarette design. Because the company representatives were in the minority, their observations were not heeded. However, it is accurate to say that manufacturing technology does not currently exist to produce, at acceptable commercial quality and rates, the experimental cigarettes that exhibited the lowest ignition propensity.

III. U.S. CIGARETTES CLAIMED TO BE "FIRE SAFE"

At one time Sherman, More and Carlton (100 Box) were touted as being the "solution". These claims were based on work done by John Krasny at NBS in 1980. The Sherman cigarettes which claimed to use no additives, come in various sizes, shapes and colors. These are specialty cigarettes which have limited distribution and should not be classified as "commercial cigarettes". The Sherman product tested by Krasny was a small circumference cigarette which employed very low porosity paper and yielded approximately 35.2 mg. of "tar". Krasny found that the Sherman he tested "self-extinguished" and did not cause ignitions. The industry position has been that the Sherman product is not "fire safe", is a non-commercial "specialty" product, and has very high deliveries.

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Krasny said More and Carlton also showed less ignition propensity on his early tests. However, they were included in the TSG test which resulted in non statistically significant differences among the 12 commercial cigarettes, as previously discussed.

The industry's basic position, now supported by the TSG work, is that there are no commercially available fire-safe cigarettes.

IV. FOREIGN CIGARETTES

Claims have also been made that certain European cigarettes were fire-safe. In the course of its research, Philip Morris has investigated cigarettes from Europe and Australia. On the whole, these cigarettes are not significantly different in their ignition propensity than cigarettes made in the United States, and Philip Morris is aware of no cigarette made anywhere in the world that could be considered "fire-safe." There are French cigarettes that were found to be less likely to ignite the mockup used in the Philip Morris tests. However, these cigarettes have a unique construction. They are made with extremely low porosity,

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yellow, corn silk paper. They have a very harsh taste and have very high deliveries. Philip Morris does not believe that these cigarettes represent a commercially acceptable alternative to the existing cigarettes sold in the United States.

V. PATENTS FOR "SELF-EXTINGUISHING" OR "FIRE-SAFE" CIGARETTES

Over the years (going back probably to the first quarter of this century) there have been over 100 patents issued for "self-extinguishing" or "fire-safe" cigarettes. Philip Morris has reviewed the patent literature and determined that, while some of the ideas contained in these patents are interesting in concept, each contains fundamental objections which prevent actual use. For example, a number of the patents call for the addition of various inorganic chemicals and other materials to tobacco or cigarette paper to make them self-extinguish. Many of these materials have known toxic effects and others are of questionable toxicity. For this reason, Philip Morris does not add these chemicals to its cigarettes.

Some patents call for the use of fire barriers mid-way down the tobacco rod to extinguish the cigarette at a given point.

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These patents fail to meet the goal of fire safety and would likely ignite a flammable substrate prior to reaching the fire barrier. Cigarettes burn slower when placed on a substrate (e.g., a couch or mattress) than they burn in air. A cigarette designed to extinguish within three to five minutes in air could burn ten to fifteen minutes on a substrate before reaching a fire barrier. By this time, most flammable substrates will have ignited. Many of these fire barrier patents call for the use of inorganic materials which are not acceptable for use in cigarettes and many also present difficult manufacturing problems. There also are obvious subjective problems with cigarettes which extinguish because of fire barriers.

Some patents would require coating the cigarette paper with various materials which reduce the porosity (permeability) of the paper and cause the cigarette to self extinguish. Most of these call for the use of unacceptable inorganic chemicals or other materials which introduce questions of toxicity. Also, the use of very non-porous paper in cigarette construction affects the efficiency with which the tobacco burns and increases deliveries of tar, nicotine and other components. This is inconsistent with the industry's efforts over the years to meet consumer demand for cigarettes with reduced deliveries. Another uncertainty with low

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porosity papers is a possible change in the composition of the smoke caused by less efficient tobacco combustion.

A number of patents call for the use of devices or gadgets that even a casual observer would have to conclude are wholly impractical from a technical standpoint. For example, some propose the insertion of a water balloon in the cigarette that would melt and quench the coal at a given point. Others would encase the cigarette in a movable metal or ceramic shield requiring the smoker to keep moving the shield to smoke the cigarette.

The Technical Study Group received samples of patented "self-extinguishing" or "fire-safe" cigarettes from five patent holders and tested them for ignition potential on a mockup test. The tests showed that these cigarettes were generally less likely to ignite certain fabrics than commercial cigarettes. The TSG did not evaluate them for technical feasibility, commercial feasibility or possible toxic effects. It concluded that "several patented approaches also offer directions for further investigation." Philip Morris has reviewed these approaches and found them to be unacceptable for the reasons stated above.

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In short, Philip Morris has reviewed the patent literature. Where the patents or ideas embodied in the patents have shown promise, Philip Morris has conducted further investigation. To date, no satisfactory solution has been found. Simply because a concept is patented does not mean it has commercial value.

VI. PHILIP MORRIS RESEARCH

In the late 70s, Philip Morris began an extensive in-house research program to investigate if cigarette construction parameters could be altered to significantly reduce the likelihood of cigarette ignition. Philip Morris discovered that the ignitability of a substrate is determined to a far greater extent by characteristics of the substrate than by the characteristics of the cigarette. On the vast majority of substrates either all cigarettes ignite or no cigarettes ignite, regardless of the construction of the cigarette. It is only in a very narrow range of fabric materials that differences among cigarettes may be found. And the ignitability of these materials is not consistent from lot-to-lot or bolt-to-bolt among fabrics made to the same specifications. These findings are consistent with those of the Technical Study Group and others who have conducted research in

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this area. Philip Morris was able to find a fabric which, within the same bolt, consistently showed differences in time to ignition when exposed to burning cigarettes of various construction parameters. For the most part, all cigarettes ignited this fabric, but in some cases there was a variance in the time necessary for ignition to take place. Some cigarettes ignited the fabric in one to two minutes, while others did not ignite the fabric for five minutes.

Using a furniture mockup made with the material described above, Philip Morris developed a program by which it experimented with differences in time to ignition from cigarettes made with various construction parameters. Philip Morris found that certain parameters could be altered to lengthen the amount of time necessary to ignite the fabric or, in some instances, cause the cigarette to self-extinguish on the mockup. However, there were a number of problems with these cigarette designs. In the first place, it is very difficult to extrapolate from differences in time to ignition on this mockup to any real world situation. A cigarette that will ignite a substrate in five minutes in a laboratory experiment, as opposed to three minutes, is still likely to start a fire in the real world if left to smolder unattended in a couch or bed. Also, the cigarette parameters that lengthened time to ignition usually

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involved reducing the porosity of paper beyond that which is acceptable from a manufacturing and consumer acceptability standpoint or adding certain chemicals to the cigarette.

In the mid-1980's, Philip Morris scientists noticed an apparent correlation between mass burn rate and cigarette ignition propensity on various types of mockup tests. Cigarettes with low mass burn rates seemed to perform better on these tests than cigarettes with high mass burn rates. Philip Morris has continued to perform research, which is highly proprietary, to develop a cigarette with a lowered mass burn rate that would be subjectively acceptable. Three research prototypes have been developed. The first two prototypes have moderately reduced mass burn rates that cannot be considered significantly less likely to start a fire, but serve as bench marks for the project. These two models did not perform as well as controls in subjective tests. The third model has a significantly reduced mass burn rate which approaches (but does not reach) the level necessary to be considered less fire prone. This model performed very poorly on subjective tests.

Research is continuing to seek an acceptable product that will not ignite furniture or bedding materials.

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VII. COMPARISON OF PENDING MOAKLEY/CRANSTON AND BOUCHER BILLS

The initial industry position with respect to legislation was that it is unnecessary and inappropriate because one cannot legislate science into existence. However, because of the potential passage of legislation in various states and other considerations, the manufacturers did not to oppose the compromised Moakley bill which became the Cigarette Safety Act of 1984 and called for a study of the problem. Armed with the 1987 TSG report, Moakley and Cranston have each introduced new legislation which requires the Secretary of Health and Human Services to consult with NBS and other agencies, consider the findings of the TSG, and within one year issue a fire safety standard for cigarettes to reduce the risk of ignition. The companies would have one year from the date the rule is issued to make cigarettes which would meet the fire safety standard. Enforcement of the standard would be under the jurisdiction of the FDA. The bills would not preempt states from prescribing standards which are more stringent than that issued by the Secretary of HHS.

This approach relies on the "quick and dirty" development of a test method and ignores issues of consumer acceptance and

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possible adverse changes in the composition of smoke from modified cigarettes. The importance of the latter two considerations was emphasized by the IAC in its report to Congress.

Development of a valid and reliable test standard is a highly complex scientific problem. Any such test must relate in a meaningful way to the broad range of fabrics and cushion materials in use in the real world. The test must also account for the wide variance among fabrics and cushion materials ordered to the same specifications from the same manufacturer. Finally, such a test should take into account the various configurations in which a carelessly handled cigarette could be dropped onto furniture and mattresses. It is unreasonable to expect that a valid, reliable test could be developed within one year. As noted, these bills fail to even mention consumer acceptance and changes in smoke composition.

On April 29, 1988, legislation was introduced by Congressman Boucher which is supported by the Tobacco Institute. This bill would continue the IAC and substitute an Implementation Task Force (ITF) for the TSG. The ITF would consist of 8 scientists from various government agencies and outside organizations with relevant expertise, and one representative from each of 5 cigarette

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manufacturers. The ITF would continue the work suggested by the TSG, i.e., attempt to develop a validated test method to determine ignition propensity, develop knowledge on changes in the composition of smoke of modified cigarettes, and assess the commercial feasibility and economic impact of manufacturing and marketing products with reduced ignition propensity. The ITF would file an interim 18-month report followed by a final report from the IAC to Congress at the end of an additional 18 months (total of 3 years).

Copies of the Moakley, Cranston and Boucher bills are available for review.

The basic position of the industry is that because of political realities, some kind of federal legislation may be necessary. The Boucher bill is hoped to be an approach which can be enacted in large part. The presence of federal legislation is important in holding off passage of "quick fix" legislation in one or more states.

VIII. STATE LEGISLATION

Potential passage of legislation in one or more states is a serious concern. Generally, the state proposals authorize a

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government official (who has no expertise) to develop an ignition performance test in a short time (usually one year) with little or no funding. The proposals usually provide the manufacturers with a one-year period in which to produce products which will meet the performance test. So far, The Tobacco Institute has been successful in the state legislatures. But the importance of federal legislation to this effort is obvious.

IX. SUMMARY

A summary position on the overall subject should continue to express concern regarding tragic accidental fires which involve the careless handling of cigarettes and other smoking materials. The industry believes that proposed solutions through modifications to the cigarette raise complex scientific questions which are not amenable to easy, quick-fix legislation. The industry has responded to the fire issue in a responsible way by supporting fire education and prevention efforts. It has cooperated fully with the efforts of the Technical Study Group and supports legislation to continue its cooperation in seeking answers to the questions that remain. Philip Morris continues its research for an acceptable product which will not ignite furniture and bedding materials.

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If there are additional questions or issues to be addressed, please contact Lee Stanford or Bill Crampton.

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RECOMMENDATIONS OF THE INTERAGENCY
COMMITTEE ON CIGARETTE AND LITTLE CIGAR FIRE SAFETY
December, 1987

SUMMARY

The Interagency Committee on Cigarette and Little Cigar Fire Safety, having reviewed the findings of its Technical Study Group, is making four recommendations that, taken together, should determine the commercial feasibility, consumer acceptance and health implications of producing cigarettes less likely to ignite mattresses and upholstered furniture. Those recommendations call for:

* development and testing of potentially feasible prototype cigarettes with reduced ignition propensity for consumer acceptability and smoke toxicity.

* development of a standardized ignition propensity test method by which the effectiveness of future "less fire-prone" cigarettes can be measured.

* assignment of this work to the National Bureau of Standards (NBS) which will either undertake, contract for, or validate the tasks in question.

* creation of a scientific, nine-member Advisory Committee to guide this research, report on its findings and make recommendations to Congress on those findings.

To encourage prompt completion of this work, the Interagency Committee also recommends that the necessary research be completed and follow-up recommendations filed within two years of the date funds are appropriated for the project. Should the ultimate result be the commercial marketing of less fire-prone cigarettes, many lives could be saved, burn injuries prevented and property damage significantly reduced in the United States each year.

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BACKGROUND

In 1984, Congress adopted the Cigarette Safety Act to determine the technical and commercial feasibility of developing cigarettes and little cigars that would be less likely to ignite upholstered furniture and mattresses. To oversee the research necessary to make these determinations, an Interagency Committee (IAC) on Cigarette and Little Cigar Fire Safety was established, consisting of the Chairman of the U.S. Consumer Product Safety Commission, the U.S. Fire Administrator and the Assistant Secretary of Health of the Department of Health and Human Services. Also, a Technical Study Group (TSG) was created to direct the research and to report to the IAC on its findings. Within 60 days of submission of that report, the IAC was to provide Congress with any recommendations it deemed appropriate.

On September 21, 1987, the TSG culminated its work on these feasibility studies by unanimously adopting a report which the IAC then forwarded to the Congress on October 29, 1987. That report concluded that "...it is technically feasible and may be commercially feasible to develop cigarettes that will have a significantly reduced propensity to ignite upholstered furniture or mattresses." This conclusion was based on eight specific findings and was followed by recommendations for additional research and the development of a standardized cigarette ignition propensity test method.

FINDINGS

The IAC, having reviewed the work of the TSG, concludes that this report makes a significant contribution to the potential development of a less fire-prone cigarette. In particular, the TSG was able to identify four cigarette characteristics which can reduce ignition propensity, those being: (1) smaller cigarette circumference, (2) lower density tobacco, (3) less porous paper and (4) reduction of the citrate addition to the paper. Likewise, the TSG determined that the per puff tar, nicotine and carbon monoxide yields associated with some experimental (non-commercial) cigarettes containing such characteristic(s) were similar to those of the best

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selling commercial cigarettes.[1] In addition, the TSG provided useful insights into various benefits and costs that might be associated with the incorporation of these features into commercially marketed cigarettes.

There are, however, a number of questions left unanswered by the TSG's final report. As the text makes clear, the TSG did not test any experimental or patented cigarettes manifesting those characteristics, for consumer acceptance.[2] Thus, it was not possible to reliably estimate the number of less fire-prone cigarettes that might be smoked which, in turn, could have an impact on both the incidence of cigarette-initiated fires and smoking-related disease. Nor, due to time constraints, was an attempt made to develop prototype cigarettes that are likely to reduce ignition propensity and achieve such acceptance. As a consequence, the TSG was not able to determine whether the smoke chemistry of such prototypes would differ in significant ways from that of cigarettes currently on the market, a consideration of paramount importance since even a small increase in the likelihood of cancer, heart attacks or lung disease would more than offset the reduction in deaths and injuries projected to result from a decrease in cigarette ignited fires. Thus, the commercial feasibility and health implications of developing a less fire-prone cigarette are undetermined and will remain that way until such time as potentially marketable varieties are developed, test marketed and analyzed for toxicity.

RECOMMENDATIONS

With that in mind, the Interagency Committee recommends that legislation be adopted to facilitate the development and testing of a less fire-prone cigarette. Specifically, the IAC suggests that it and the TSG be replaced by a new, nine member Advisory Committee which, after completion of the necessary technical work, would submit a final report to the Congress, complete with findings and recommendations, on the commercial feasibility and health implications

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[1] "Toward a Less Fire-Prone Cigarette," Final Report of the Technical Study Group on Cigarette and Little Cigar Fire Safety, October, 1987, p. 15.

[2] Ibid., p. 11.

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of producing such a cigarette. This Advisory Committee would provide guidance to and receive administrative support from the National Bureau of Standards (NBS) which, in turn, would undertake, contract for, or validate prototype development, consumer acceptance testing (which the IAC believes is more properly the role of industry), smoke chemistry analysis (to determine potential short and long term health effects) and the creation of a standardized ignition propensity performance test method. Should prototype testing support development and eventual marketing of cigarettes with reduced ignition propensity, the existence of such a test method would provide a prompt, dependable way of determining whether cigarettes of that description lived up to expectations.

Relying on its experience over the past three years, the IAC estimates that the cost, to the federal government, of these projects would be approximately:

(1)	Consumer acceptance testing/ validation	-- $50,000
(2)	Smoke chemistry analysis (from existing sources, if appropriate)	-- 50,000
(3)	Development of a standardized ignition propensity test method	-- 300,000
(4)	Advisory Committee travel expenses (based on a nine member Committee holding four meetings per year for two years)	-- 100,000
	Total for two years	$500,000

As for the actual production of prototype cigarettes, the IAC recommends that consideration be given to asking the industry to donate, to the Advisory Committee (AC), the prototype cigarettes needed for testing. Not only was a similar arrangement of considerable benefit to the TSG during the recently completed cigarette ignition propensity work but, as a practical matter, obtaining prototypes from other sources could be more difficult and time consuming.

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Based on these estimates, the IAC recommends that the new AC be given two years to complete its work from the time funds are appropriated for that purpose. During that time, the AC should provide periodic progress reports to Congress. In addition, the IAC recommends that a total of $500,000 be appropriated to the NBS in support of this effort. Of this sum, the IAC suggests that $100,000 should be specifically earmarked for travel and expenses associated with Advisory Committee meetings.

With respect to the specific makeup of this Advisory Committee, the IAC observes that considerable scientific expertise will be required for it to successfully carry out its responsibilities. Therefore, the IAC suggests that this entity be comprised of individuals with extensive technical background(s) in public health, cigarette production, smoke toxicity testing, test method development and/or market survey techniques. Also, the IAC recommends that the meetings of this Advisory Committee be open to the public, except when trade secret or proprietary information is being discussed, and that the public be notified in advance of the dates of these meetings.

CONCLUSION

In making these recommendations, the Interagency Committee is acutely aware of what is at stake. If additional research leads to the successful development of a cigarette less likely to start furniture fires, up to an estimated 1,500 deaths, 7,000 injuries and nearly $500 million in property losses could be prevented in the United States each year.[3] However, the IAC has no desire to subject the American taxpayer to the burden of financing an interminable series of studies that never resolve the basic issue of feasibility. Indeed, extended delay in the marketing of a less fire-prone cigarette, should such a product be feasible, would deprive the American public of much of the benefit that could otherwise result from its development. Thus, the IAC believes the issues of feasibility, consumer acceptance and health implications need to be resolved one way or the other as expeditiously as good science will allow. And it firmly believes these recommendations will result in just such a resolution.

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[3] Ibid p. 3.

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CREDITS

The Interagency Committee wishes to thank the members of the Technical Study Group (TSG), the appropriate staff of the National Bureau of Standards and the individual contractors who conducted and analyzed the technical work upon which these recommendations are based. As noted in its "Findings," the TSG significantly advanced the state of knowledge in the area of cigarette ignition propensity and deserves particular credit for having done so.

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U.S. CONSUMER PRODUCT SAFETY COMMISSION

U.S. FIRE ADMINISTRATION

U.S. PUBLIC HEALTH SERVICE

INTERAGENCY COMMITTEE ON CIGARETTE AND LITTLE CIGAR FIRE SAFETY

Release: December 23, 1987

FEDERAL PANEL CALLS FOR FURTHER WORK ON
CIGARETTES LESS LIKELY TO CAUSE FIRES

WASHINGTON, DC -- A federal panel has recommended that Congress authorize additional research into the commercial feasibility and health implications of cigarettes which are less likely to ignite upholstered furniture and mattresses. Should this research lead to the successful development of a less fire-prone cigarette as many as 1,500 deaths, 7,000 serious burn injuries and nearly $500 million in property losses could be prevented in the United States each year.

Authoring these recommendations was the Interagency Committee on Cigarette and Little Cigar Safety, consisting of Mr. Terrence Scanlon, Chairman of the U. S. Consumer Product Safety Commission; Mr. Clyde Bragdon, the U. S. Fire Administrator and; Dr. Robert Windom, Assistant Secretary for Health of the U. S. Department of Health and Human Services. Its findings were based on a three-year technical study, completed several months ago, which concluded that "... it is technically feasible and may be commercially feasible to develop cigarettes that will have a significantly reduced propensity to ignite upholstered furniture and mattresses."

As a preface to its recommendations, the three member government panel commended the technical study for identifying four cigarette characteristics which can reduce the likelihood of ignition: (1) smaller cigarette circumference, (2) lower density tobacco, (3) less porous paper and (4) reduction of the citrate addition to cigarette paper. The panel also noted that the technical study had determined that the per puff tar, nicotine and carbon monoxide yields associated with some experimental cigarettes having such characteristics were similar to those of the best selling commercial cigarettes.

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But, according to the Interagency Committee, additional testing is needed to determine the marketability of less fire-prone cigarettes plus any increased health risks smokers might incur from such cigarettes. To accomplish this, the panel recommended that Congress establish a nine member Advisory Committee of scientists. This Advisory Committee, if approved by Congress, would guide the work of the Commerce Department's National Bureau of Standards which would undertake or contract for prototype cigarette development, consumer acceptance testing/validation, smoke chemistry analysis and the creation of a standardized ignition propensity performance test method. Once that work was completed, which is expected to take two years, the Advisory Committee would report to Congress on its findings and recommendations.

Copies of the Interagency Committee recommendations to Congress and the technical study on which they are based can be obtained by writing the Secretariat, Interagency Committee on Cigarette and Little Cigar Fire Safety, Consumer Product Safety Commission, Room 420, Washington, DC 20207 (telephone 301-492-6554). For any additional information contact the Office of Media Relations, Consumer Product Safety Commission, Washington, DC 20207 (telephone 301-492-5540).

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TOWARD A LESS FIRE-PRONE CIGARETTE

FINAL REPORT

OF THE

TECHNICAL STUDY GROUP ON CIGARETTE
AND LITTLE CIGAR FIRE SAFETY

CIGARETTE SAFETY ACT OF 1984

September 21, 1987

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EXECUTIVE SUMMARY

The Cigarette Safety Act of 1984 created the Technical Study Group on Cigarette and Little Cigar Fire Safety (TSG), directing it to "undertake such studies and other activities as it considers necessary and appropriate to determine the technical and commercial feasibility, economic impact, and other consequences of developing cigarettes and little cigars that will have a minimum propensity to ignite upholstered furniture or mattresses." Lower ignition-prone cigarettes would significantly reduce fire losses. Since its formation in January, 1985, the TSG has carried out a program of consultation, deliberation, and research. This text is the final report on that program.

The Technical Study Group finds that it is technically feasible and may be commercially feasible to develop cigarettes that will have a significantly reduced propensity to ignite upholstered furniture or mattresses. Furthermore, the overall impact on other aspects of the United States society and economy may be minimal. Thus it may be possible to solve this problem at costs that are less than the potential benefits, assuming the commercial feasibility of the modified cigarettes.

The particular conclusions of this study are as follows:

- There are cigarette characteristics whose variations in the laboratory reduced the ignition propensity of the cigarette. These are: reduced circumference, lower density tobacco, less porous paper, and reduction of citrate addition to the paper. Considerably larger reductions were achieved with combinations of these. Several patented approaches also offer directions for further investigation. Limited evidence suggests the presence of a filter may also have some effect on ignition propensity.

- The differences in ignition propensity among selected current commercial cigarettes are unimportant.

- Measurements of cigarette ignition propensity on upholstered furniture mockups are reasonable indicators of performance on full-scale furniture made of the same materials. However, the wide lot-to-lot variation in those materials limits the use of such mockups for cigarette testing over a long period of time and by different laboratories.

- A valid and reliable measurement method is needed to determine that a cigarette is less ignition-prone. It is also important to collect information on cigarette-initiated fires to determine how successfully future cigarettes perform.

- The manufacture of less fire-prone cigarettes may require some advances in cigarette design and manufacturing technology.

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- No cigarettes were tested for their acceptability to the smoking public. However, some physical characteristics which decrease a cigarette's propensity to ignite soft furnishings are incorporated individually in some commercial cigarettes. Combinations of all characteristics of the lowest ignition propensity cigarettes have not been incorporated in any commercial cigarettes.

- The overall effects of the cigarette modifications considered may result in only small changes in the price of cigarettes, unemployment, health care costs, life expectancy, and the financial status of the affected industries and professions. This conclusion involves a number of broad but necessary assumptions.

- The per puff tar, nicotine, and carbon monoxide yields from some of the least ignition-prone experimental cigarettes were within the ranges of yields from the best-selling commercial cigarettes. These cigarettes have a significantly higher resistance to puffing and a marginally lower number of puffs than do current commercial cigarettes. The toxicity of smoke from a future low ignition propensity cigarette needs to be addressed, as would the smoke from any substantially modified commercial cigarette, before its introduction into the marketplace.

The Technical Study Group recommends the following technical activities be pursued:

- A standard test method is needed to determine cigarette ignition propensity. Such a method should be developed as soon as possible and validated using the current set of experimental cigarettes.

- Performance data for current market cigarettes should be generated using the new test method. These data could then be compared to future year cigarette performance.

- A study to collect baseline and follow-up data about the characteristics of cigarettes, products ignited, and smokers involved in fires should be designed and implemented.

- Systematic knowledge should be developed (from existing or new sources, as appropriate) on (a) changes in the toxicity of smoke and resultant health effects from modified cigarettes, and (b) societal costs of injuries from cigarette-initiated fires.

- Both the laboratory studies on and computer modeling of ignition physics should be continued to develop a valid, user-friendly predictive capability. This would enable inexpensive screening for ignition propensity of future cigarette concepts. (The authors of reference 19 have offered more detailed recommendations for further research.)

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Table 1. Description of Experimental Cigarettes

No.	Tobacco	Tobacco Density	Paper Porosity	Citrate Added	Circumference (mm)	Second Paper Wrapping[c]
101	Burley	High	Low	Yes	21	No
102	Burley	High	Low	No	21	No
103	Burley	High	High	Yes	21	No
104	Burley	High	High	No	21	No
105	Burley	Low	Low	Yes	21	No
106	Burley	Low	Low	No	21	No
107	Burley	Low	High	Yes	21	No
108	Burley	Low	High	No	21	No
109	Flue-Cured	High	Low	Yes	21	No
110	Flue-Cured	High	Low	No	21	No
111	Flue-Cured	High	High	Yes	21	No
112	Flue-Cured	High	High	No	21	No
113	Flue-Cured	Low	Low	Yes	21	No
114	Flue-Cured	Low	Low	No	21	No
115	Flue-Cured	Low	High	Yes	21	No
116	Flue-Cured	Low	High	No	21	No
117	Burley	High	Low	Yes	25	No
118	Burley	High	Low	No	25	No
119	Burley	High	High	Yes	25	No
120	Burley	High	High	No	25	No
121	Burley	Low	Low	Yes	25	No
122	Burley	Low	Low	No	25	No
123	Burley	Low	High	Yes	25	No
124	Burley	Low	High	No	25	No
125	Flue-Cured	High	Low	Yes	25	No
126	Flue-Cured	High	Low	No	25	No
127	Flue-Cured	High	High	Yes	25	No
128	Flue-Cured	High	High	No	25	No
129	Flue-Cured	Low	Low	Yes	25	No
130	Flue-Cured	Low	Low	No	25	No
131	Flue-Cured	Low	High	Yes	25	No
132	Flue-Cured	Low	High	No	25	No
201	Flue-Cured	Low	Very Low	No	21	No
202	Flue-Cured	Low	Very Low	No	25	No
203	Flue-Cured	Low	Very Low[a]	No	25	No
204	Flue-Cured	Low	Very Low[b]	No	25	No
205	Flue-Cured	Low	Very Low[a,b]	No	25	No
206	Flue-Cured	Low	Very Low	No	25	Yes
207	Flue-Cured	Low	Very Low[a]	No	25	Yes
208	Flue-Cured	Low	Very Low[b]	No	25	Yes
209	Flue-Cured	Low	Very Low[a,b]	No	25	Yes

[a] Electrostatically perforated to "high" permeability after manufacture.
[b] Embossed (to separate the burning tobacco from the substrate)
[c] Two paper layers; inner wrap is extremely porous.

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Source: https://www.industrydocuments.ucsf.edu/docs/rhbj0000

Table 2. Ignition Propensities of Selected Test Cigarettes [19]

	Designation	No. Ignitions in 20 Tests	%
Experimental Cigarettes	201	0	0
	106	1	5
	202	2	10
	130	4	20
	114	4	20
	105	6	30
	113	6	30
	108	7	35
	122	7	35
	129	10	50
	107	11	55
	120	20	100
	127	20	100
Least Ignition-Prone Commercial Cigarettes	2	12	60
	1	16	80
Typical Ignition Propensity Commercial Cigarettes	3	18	90
	6	20	100

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CONFIDENTIAL

PHILIP MORRIS
U.S.A.
RESEARCH CENTER: P.O. BOX 26583, RICHMOND, VIRGINIA 23261-6583 TELEPHONE (804) 274-2000

November 10, 1987

Mr. Colin B. Church
Consumer Product Safety Commission
Room 420
Washington, D.C. 20207

RE: **Recommendations to the Interagency Committee**

Dear Colin:

I understand that the Interagency Committee is in the process of compiling information for submission of policy recommendations to Congress on cigarette fire safety. Thank you for the opportunity to contribute my views on this important subject.

As a member of the Technical Study Group (TSG), I support the conclusions and recommendations for further work which are set forth in the final report of the TSG. I will not attempt to restate these conclusions and recommendations, but I would like to emphasize certain areas of work which need to be completed in order to insure fulfillment of the progress which has been achieved so far. Specifically, I view the following to be of special importance:

(1) Validated Test Method: Perhaps the first order of business should be the development of a validated test method which will reliably measure the ignition propensity of cigarettes on soft furnishings. The task will not be easy and it is complicated by the lack of uniformity among fabrics and filling materials, e.g., cotton batting and polyurethane foams. It is critical that a reproducible, validated test be developed which has relevance to furnishings in the real world. In this regard the test should take into account the effects of furniture construction factors such as, multiple layers of fabrics as occur in cushion seams, welt cords and surface firmness, i.e., the extent to which the fabric is stretched over filling materials.

(2) Analysis of Furnishings Currently in Use: It is important to determine if the test methodology is relevant, that is, the extent to which test materials are representative of furnishings existing in the real world. Therefore, I recommend an analysis of fabrics and filling materials currently in use to permit realistic extrapolations from test results to real world circumstances.

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(3) Scientific Evaluation of Changes in Smoke Composition: The final report identified certain cigarette parameters which, in combination, resulted in reduced cigarette ignition propensity. One of the most significant of these parameters, low paper porosity, may result in changes in smoke composition. Such changes must be determined and evaluated by appropriate scientific analysis before a major modification in paper permeability is employed.

(4) Acceptance By the Consumer: The cigarettes identified in the TSG report as having the lowest ignition propensity were made experimentally at the limits of manufacturing technology. They are well outside the range of products in the market and possess a number of characteristics that will undoubtedly cause them to be rejected by the smoking public. Although the TSG did not have the time needed to look at the question of consumer acceptance, that critical issue must be addressed as work in this area continues on any additional test products.

(5) Consideration of Substrate Materials: It is a well-known fact that the fabric and filling materials onto which a carelessly-handled cigarette may fall is a determinative factor in whether an ignition occurs. The TSG found a wide lot-to-lot variation among materials ordered from the same manufacturer. In addition, the metal ion content of these materials is of critical significance. Knowledge of the effects of these factors is of vital importance in achieving solutions to careless fires.

(6) Scientific Expertise: Because most of the matters to be addressed are primarily scientific, I believe the group selected to investigate these issues should be composed of persons with appropriate scientific expertise.

It is obvious from the foregoing that I believe work remains to be completed before real solutions are reached. The issues are complex, but significant progress has been made. It is my hope that the points I have discussed and the recommendations contained in the TSG report be addressed in an objective and scientific manner. I pledge my continued cooperation and efforts in this regard.

Sincerely,

James L. Charles, Ph. D.
Director, Research

/nb

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100TH CONGRESS
1ST SESSION

H. R. 3440

To direct the Secretary of Health and Human Services to promulgate fire safety standards for cigarettes and for other purposes.

IN THE HOUSE OF REPRESENTATIVES

OCTOBER 7, 1987

Mr. MOAKLEY (for himself, Mr. WAXMAN, Mr. WHITTAKER, Mr. BIAGGI, Mrs. BOXER, and Mr. CONYERS) introduced the following bill; which was referred to the Committee on Energy and Commerce

---

A BILL

To direct the Secretary of Health and Human Services to promulgate fire safety standards for cigarettes and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled,

SECTION 1. SHORT TITLE, FINDINGS.

(a) SHORT TITLE.—This Act may be cited as "Fire Safe Cigarette Act of 1987".

(b) FINDINGS.—The Congress finds that—

(1) cigarette ignited fires are the leading cause of fire deaths in the United States,

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(2) in 1984 there were 1,570 deaths from cigarette ignited fires, 7,000 serious injuries from such fires, and $390 million in property damage caused by such fires,

(3) the final report of the Technical Study Group on Cigarette and Little Cigar Fire Safety under the Cigarette Safety Act of 1984 has determined that it is technically feasible to produce a cigarette with less propensity to ignite fires,

(4) it is appropriate for the Congress to require by law the establishment of a fire safety standard for the manufacture and importation of cigarettes,

(5) the value of a fire safety standard which will prevent property damage and personal injury and loss of life is economically incalculable, and

(6) it is appropriate that the regulatory expertise of the Food and Drug Administration be used to implement a fire safety standard.

SEC. 2. FIRE SAFETY STANDARD.

(a) IN GENERAL.—Not later than one year after the date of the enactment of this Act, the Secretary shall by rule issue a fire safety standard for cigarettes to reduce the risk of ignition presented by cigarettes. In establishing the standard the Secretary shall—

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(1) consult with the National Bureau of Standards and seek the advice and expertise of other Federal and State agencies engaged in fire safety, and

(2) take into account the findings in the report of the Technical Study Group on Cigarette and Little Cigar Fire Safety under the Cigarette Safety Act of 1984 (15 U.S.C. 2054 note).

(b) STOCKPILING.—The Secretary shall include in the rule issued under subsection (a) a prohibition of stockpiling of cigarettes to which the standard issued under subsection (a) will apply. For purposes of this subsection, the term "stockpile" means the manufacturing or importing of a cigarette between the date a standard is issued under subsection (a) and the date the standard is to take effect at a rate greater than the rate the cigarettes were manufactured or imported for the one year period ending on the date the standard was issued.

(c) PROCEDURE.—The rule under subsection (a) shall be issued—

(1) in accordance with section 553 of title 5, United States Code, except that if the rule is significantly different from the recommendation of the Technical Study Group on Cigarette and Little Cigar Fire Safety under the Cigarette Safety Act of 1984 the Secretary shall include in the statement under subsec-

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tion (c) of such section an explanation for the difference, and

(2) without regard for the requirements of Executive Order 12291.

(d) EFFECTIVE DATE.—The Secretary shall prescribe the effective date of the rule issued under subsection (a), except that such date may not be later than 2 years after the date of the enactment of this Act.

(e) JUDICIAL REVIEW.—

(1) Any person who is adversely affected by a rule issued under subsection (a) may, at any time before the 60th day after the Secretary issues the rule, file a petition with the United States Court of Appeals for the District of Columbia Circuit or for any other circuit in which such person resides or has its principal place of business to obtain judicial review of the rule. A copy of the petition shall be forthwith transmitted by the clerk of the court to the Secretary. The Secretary shall file in the court the record of the proceedings on which the Secretary based the rule as provided in section 2112 of title 28, United States Code.

(2) If the petitioner applies to the court for leave to adduce additional evidence, and shows to the satisfaction of the court that such additional evidence is material and that there was no opportunity to adduce

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such evidence in the proceeding before the Secretary, the court may order such additional evidence (and evidence in rebuttal thereof) to be taken before the Secretary in a hearing or in such other manner, and upon such terms and conditions, as the court deems proper. The Secretary may modify the Secretary's findings as to the facts, or make new findings, by reason of the additional evidence so taken, and the Secretary shall file such modified or new findings, and the Secretary's recommendations, if any, for the modification of the rule.

(3) Upon the filing of a petition under paragraph (1), the court shall have jurisdiction to review the rule of the Secretary, as modified, in accordance with chapter 7 of title 5, United States Code.

(f) IMPLEMENTATION.—The Secretary shall use the Food and Drug Administration to carry out this Act.

SEC. 3. ENFORCEMENT.

(a) PROHIBITION.—No person may manufacture or import a cigarette unless the cigarette is in compliance with a fire safety standard issued under section 2(a).

(b) PENALTY.—A violation of subsection (a) shall be considered a violation of section 301 of the Federal Food, Drug, and Cosmetic Act.

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SEC. 4. PREEMPTION.

(a) IN GENERAL.—This Act and the fire safety standard promulgated under section 2(a) do not preempt or otherwise affect in any way any law of a State or political subdivision which prescribes a fire safety standard for cigarettes which is more stringent than the standard promulgated under section 2(a).

(b) DEFENSES.—In any civil action for damages compliance with the fire safety standard promulgated under section 2(a) may not be admitted as a defense.

SEC. 5. DEFINITIONS.

For purposes of this Act:

(1) The term "Secretary" means the Secretary of Health and Human Services.

(2) The term "cigarette" has the meaning prescribed by section 3 of the Federal Cigarette Labeling and Advertising Act.

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Source: https://www.industrydocuments.ucsf.edu/docs/rhbj0000

II

100TH CONGRESS
1ST SESSION

S. 1763

To direct the Secretary of Health and Human Services to promulgate fire safety standards for cigarettes and for other purposes.

IN THE SENATE OF THE UNITED STATES

OCTOBER 7 (legislative day, SEPTEMBER 25), 1987

Mr. CRANSTON (for himself, Mr. HEINZ, Mr. STAFFORD, and Mr. BOSCHWITZ) introduced the following bill; which was read twice and referred to the Committee on Commerce, Science, and Transportation

---

A BILL

To direct the Secretary of Health and Human Services to promulgate fire safety standards for cigarettes and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled,

SECTION 1. SHORT TITLE.

This Act may be cited as "An Act to Reduce Fires Caused by Cigarettes".

SEC. 2. DEFINITIONS.

For purposes of this Act—

(1) SECRETARY.—The term "Secretary" means the Secretary of Health and Human Services.

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(2) CIGARETTE.—The term "cigarette" has the meaning prescribed by section 3 of the Federal Cigarette Labeling and Advertising Act.

SEC. 3. FIRE SAFETY STANDARDS.

(a) IN GENERAL.—Not later than 1 year after the date of the enactment of this Act, the Secretary shall by rule issue a fire safety standard for cigarettes to reduce the risk of ignition presented by cigarettes.

(b) CRITERIA.—In establishing the standard the Secretary shall—

(1) consult with the National Bureau of Standards and seek the advice and expertise of other Federal and State agencies engaged in fire safety; and

(2) take into account the findings in the report of the Technical Study Group on Cigarette and Little Cigar Fire Safety under the Cigarette Safety Act of 1974 (15 U.S.C. 2054 note).

(c) PROCEDURE.—The rule under subsection (a) shall be issued—

(1) in accordance with section 553 of title 5, United States Code, except that if the rule is significantly different from the recommendations of the Technical Study Group on Cigarette and Little Cigar Fire Safety pursuant to the Cigarette Safety Act of 1984 (15 U.S.C. 2054 note), the Secretary shall include in

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the statement required under subsection (c) of section 553, an explanation of the reasons for the difference; and

.(2) without regard for the requirements of Executive Order 12291.

(d) EFFECTIVE DATE.—The Secretary shall prescribe the effective date of the standard issued under subsection (a), except that such date may not be later than 2 years after the date of the enactment of this Act.

(e) STOCKPILING.—

(1) IN GENERAL.—The Secretary shall prohibit a manufacturer of cigarettes from stockpiling cigarettes that are subject to the standards issued under subsection (a).

(2) DEFINITION.—For purposes of this subsection, the term "stockpile" means the manufacturing or importing of a cigarette between the date a standard is issued under subsection (a) and the date the standard is to take effect, at a rate greater than the rate the cigarettes were manufactured or imported for the 1 year period ending on the date the standard was issued.

(f) JUDICIAL REVIEW.—

(1) COURT OF APPEALS.—

(A) IN GENERAL.—Any person who is adversely affected by the standard issued under sub-

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section (a) may, at any time before the 60th day after the Secretary issues the standard, file a petition with the United States Court of Appeals for the District of Columbia Circuit, or for any other circuit in which such person resides or has its principal place of business, to obtain judicial review of the standard.

(B) NOTIFICATION OF SECRETARY.—A copy of the petition filed under subparagraph (A) shall be promptly transmitted by the clerk of the court to the Secretary.

(C) RECORD OF PROCEEDINGS.—The Secretary shall file in the court the record of the proceedings on which the Secretary based the standard as provided in section 2112 of title 28, United States Code.

(2) ADDITIONAL EVIDENCE.—

(A) HEARING.—If the petitioner applies to the court for leave to discover additional evidence, and shows to the satisfaction of the court that such additional evidence is material and that there was no opportunity to discover such evidence in the proceeding before the Secretary, the court may order such additional evidence (and evidence in rebuttal thereof) to be taken before the Secre-

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tary in a hearing or in such other manner, and on such terms and conditions, as the court deems proper.

(B) MODIFICATION OF FINDINGS.—The Secretary may modify the prior findings as to the facts, or make new findings, by reason of the additional evidence so taken, and the Secretary shall file such modified or new findings, and the recommendations of the Secretary, if any, for the modification of the standard.

(3) REVIEW OF STANDARDS.—On the filing of a petition under paragraph (1), the court shall have jurisdiction to review the standard of the Secretary, as modified, in accordance with chapter 7 of title 5, United States Code.

(g) IMPLEMENTATION.—The Secretary may carry out this Act through the Food and Drug Administration.

SEC. 4. ENFORCEMENT.

(a) PROHIBITION.—No person may manufacture or import a cigarette unless the cigarette is in compliance with a fire safety standard issued under section 3(a).

(b) PENALTY.—A violation of subsection (a) shall be considered a violation of section 301 of the Federal Food, Drug, and Cosmetic Act (21 U.S.C. 331).

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SEC. 5. PREEMPTION.

This Act does not preempt any law of a State or political subdivision of a State that prescribes a fire safety standard for cigarettes that are more stringent than the standard prescribed under section 3(a).

SEC. 6. DEFENSES.

In any civil action for damages, compliance with the fire safety standard promulgated under section 3(a) may not be admitted as a defense.

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100TH CONGRESS
2D SESSION

H. R. 4497

To implement the recommendations of the Interagency Committee and the Technical Study Group on Cigarette and Little Cigar Fire Safety, and for other purposes.

IN THE HOUSE OF REPRESENTATIVES

APRIL 29, 1988

Mr. BOUCHER (for himself, Mr. COOPER, Mr. TAUZIN, Mr. BILEY, and Mr. CALLAHAN) introduced the following bill; which was referred to the Committee on Energy and Commerce

---

A BILL

To implement the recommendations of the Interagency Committee and the Technical Study Group on Cigarette and Little Cigar Fire Safety, and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled,

SECTION 1. SHORT TITLE.

(a) SHORT TITLE.—This Act may be cited as the "Fire Safe Cigarette Implementation Act of 1988".

(b) FINDINGS.—The Congress finds that—

(1) the Interagency Committee and the Technical Study Group on Cigarette and Little Cigar Fire Safety have submitted reports to the Congress, as required by

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the Cigarette Safety Act of 1984, on the technical and commercial feasibility, economic impact, and other consequences of developing cigarettes and little cigars having a minimum propensity to ignite upholstered furniture and mattresses,

(2) those reports indicate that the Technical Study Group, during the period specified by the Congress, made substantial progress in its work and urge that the Federal effort with respect to cigarette and little cigar fire safety be continued in certain specified areas, and

(3) there is a need for Federal legislation to complete the work mandated by the Cigarette Safety Act of 1984, implement the recommendations of the Technical Study Group, and address other issues.

SEC. 2. INTERAGENCY COMMITTEE AND IMPLEMENTATION TASK FORCE.

(a) INTERAGENCY COMMITTEE.—The Interagency Committee on Cigarette and Little Cigar Fire Safety (hereinafter referred to as the "Interagency Committee"), created by the Cigarette Safety Act of 1984, is reestablished for the purpose of overseeing and reviewing the work of the Implementation Task Force (hereinafter referred to as the "Task Force") established under subsection (b). The Interagency Committee may request the head of any Federal department or agency to assist the Interagency Committee or the Task

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Force, or both, in carrying out their responsibilities. The Interagency Committee also may retain or contract with such consultants as may be deemed necessary without regard to section 3709 of the Revised Statutes of the United States (41 U.S.C. 5) and the Act of June 10, 1936 (known as the Walsh-Healey Act) (41 U.S.C. 35–45). The authority of the Interagency Committee to enter into contracts shall be effective for any fiscal year only to such extent or in such amounts as are provided in advance by appropriation Acts.

(b) IMPLEMENTATION TASK FORCE.—There is established an Implementation Task Force which shall consist of—

(1) 2 scientists having expertise in the development of a valid and reliable method for testing the ignition propensity of cigarettes and little cigars, one of whom shall be appointed by the Chairman of the Consumer Product Safety Commission from among the employees of the Commission and the other of whom shall be appointed by the Director of the Center for Fire Research of the National Bureau of Standards from among the employees of the National Bureau of Standards,

(2) 2 scientists having expertise concerning the possible consequences for smokers of any cigarette or little cigar modification, one of whom shall be appoint-

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ed by the Director of the National Institutes of Health from among the employees of the National Institutes of Health and the other of whom shall be appointed by the Chairman of the Interagency Committee, with the advice and consent of the Interagency Committee, from a list of individuals submitted by the American Health Foundation,

(3) 2 scientists having expertise in the chemistry and physics of combustion to be appointed by the Chairman of the Interagency Committee, with the advice and consent of the Interagency Committee, from lists of individuals submitted by the International Association of Fire Chiefs and the International Association of Fire Fighters,

(4) 2 scientists having expertise in the content and characteristics of soft furnishings and fabrics to be appointed by the Chairman of the Interagency Committee, with the advice and consent of the Interagency Committee, from lists of individuals submitted by the American Furniture Manufacturers Association and the Business and Institutional Furniture Manufacturers Association, and

(5) 5 scientists having expertise in the manufacture and design of cigarettes and little cigars to be appointed by the Chairman of the Interagency Commit-

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tee, with the advice and consent of the Interagency Committee, from a list of individuals submitted by The Tobacco Institute.

The persons appointed to serve on the Task Force may designate, with the advice and consent of the Interagency Committee, from among their number such persons to serve as team leaders, coordinators, or chairpersons as they deem necessary or appropriate to carry out the Task Force's functions under section 3.

SEC. 3. RESPONSIBILITIES, REPORTING.

(a) TASK FORCE RESPONSIBILITIES.—The Task Force, subject to oversight and review by the Interagency Committee, shall continue the work specified by the Cigarette Safety Act of 1984, including implementing the recommendations contained in the final report of the Technical Study Group on Cigarette and Little Cigar Fire Safety. Such activities shall include—

(1) developing a validated test method to determine the ignition propensity of cigarettes and little cigars when in contact with upholstered furniture or mattresses,

(2) developing representative performance data for current market cigarettes, using the validated test method described in paragraph (1), to serve as a baseline to compare future cigarette performance,

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(3) continuing laboratory studies on, and computer modeling of, ignition physics relating to cigarettes, little cigars, soft furnishings, and fabrics,

(4) designing and implementing a study or studies to collect baseline and follow-up data about the characteristics of cigarettes and little cigars, products ignited, and smokers involved in cigarette and little cigar fires,

(5) developing systematic knowledge (from existing or new sources as appropriate) on changes in the composition of smoke of modified cigarettes and little cigars and societal costs of injuries stemming from fires involving cigarettes or little cigars, and

(6) assessing the commercial feasibility and economic impact of manufacturing and marketing cigarettes and little cigars with reduced ignition propensity.

(b) HEARINGS.—For purposes of carrying out the functions described in subsection (a), the Interagency Committee and the Task Force may hold such hearings, sit and act at such times and places, take such testimony, and receive such evidence as the Interagency Committee or the Task Force deems appropriate.

(c) CONFIDENTIALITY.—

(1) Any information provided to the Interagency Committee or to the Task Force under subsection (a) that is designated as trade secret or confidential infor-

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mation shall be treated as trade secret or confidential information subject to section 552(b)(4) of title 5, United States Code, and shall not be revealed except as provided by paragraph (2). No member of the Interagency Committee or Task Force, and no person assigned to or consulting with such entities, shall disclose any such information to any person who is not a member of, assigned to, or consulting with the Interagency Committee or Task Force unless the person submitting such information specifically and in writing authorizes such disclosure.

(2) Paragraph (1) does not authorize the withholding of any information from any duly authorized committee or subcommittee of the Congress, except that if a committee or subcommittee of the Congress requests access to confidential information that has been submitted to the Interagency Committee or the Task Force, the Chairman of the Interagency Committee shall promptly notify the person who provided the information of the request and shall not disclose the information until ten days have elapsed from such person's receipt of written notice that the information is to be disclosed.

(3) The Interagency Committee shall adopt, on a vote of a majority of its members, reasonable proce-

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dures to protect the confidentiality of trade secret and confidential information as defined in this subsection and shall be responsible for ensuring that the Task Force follows such procedures in its handling of trade secret and confidential information.

SEC. 4. DEFINITIONS.

As used in this Act, the terms "cigarettes" and "little cigars" shall have the meanings given such terms by section 3 of the Federal Cigarette Labeling and Advertising Act.

SEC. 5. REPORTS, TERMINATION.

The Interagency Committee shall submit to the Congress 18 months after the date of enactment and funding of this Act a report prepared by the Task Force describing the Task Force's progress in carrying out the work described in section 3. The Interagency Committee shall submit to the Congress no later than 18 months thereafter a final study report prepared by the Task Force. The Interagency Committee shall provide to the Congress, within 60 days after the submission by the Interagency Committee of the Task Force's final report, any policy recommendations the Interagency Committee may deem appropriate. The Interagency Committee and Task Force shall terminate one month after the submission of such policy recommendations.

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PRIVILEGED AND CONFIDENTIAL
ATTORNEY WORK PRODUCT

RELATIONSHIP BETWEEN INDUSTRY EXPENDITURES ON ADVERTISING AND INDUSTRY EXPENDITURES ON RESEARCH

During the course of the Cipollone trial, the plaintiff argued that the tremendous disparity between annual industry expenditures on advertising and annual industry expenditures on research into the health effects of smoking was "further evidence" of the industry's "callous disregard" for the health of its consumers. It is likely that this theme may be picked up by questioners if a company executive appears as a witness before a congressional hearing. Without going into the specific dollar amounts involved, the witness may simply acknowledge the disparity (while, of course, disclaiming knowledge of precise amounts spent by competitors) and offer the following explanation:

These two categories of expenditures really cannot be compared in a meaningful way because of the great differences between the goals of advertising and research and between the available means for achieving those goals. Advertising is a recognized commercial technique followed by all manufacturers in their marketing efforts. In the case of cigarette manufacturers, advertising expenditures are affected by two special considerations that are not relevant to most other manufacturers. First, we are engaged in intense brand competition in a declining market. Thus, it is necessary for us

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to induce smokers of other manufacturer's products to switch to our brands. Naturally, our advertising expenditures under these circumstances are high. This consideration is compounded by the second special circumstance for cigarette advertising. We are limited to what advertising people refer to as "less efficient" media because television advertising has been banned. The effect of this restriction is to require many more dollars to achieve the same degree of brand recognition among smokers through the print media.

Nonetheless, our advertising by its very nature is designed to reach vast numbers of people (there are over 55 million smokers in America). The space available for our advertising in magazines, newspapers, and on billboards is also quite large. Therefore, "more advertising," reaching "more smokers," can be achieved simply through the expenditure of "more money".

The goals of research into the health effects of smoking, and the means that can be used to achieve those goals, are quite different. The companies give money for research in the hopes of trying to come closer to some of the answers in the smoking and health controversy. The object of our commitment is to obtain relevant and meritorious research.

Annual expenditures on research, thus, are limited first, by the number of high quality research proposals that are presented to the Council for Tobacco Research or to the

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individual companies annually, and second, by the number of high quality researchers who are available. "More relevant and meritorious research," therefore, is not simply a function of "more money". The additional expenditures would be wasted without the projects and personnel to put that money to use.

Thus, it should come as no surprise that there is a disparity between annual expenditures in these two categories. There is nothing insidious in that disparity. It is simply a recognition and expression of the differences between the ways and means of advertising and of research.

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PRIVILEGED AND CONFIDENTIAL
ATTORNEY WORK PRODUCT

ENVIRONMENTAL TOBACCO SMOKE

Environmental tobacco smoke (ETS), also sometimes referred to as "passive (or involuntary) smoking", is a shorthand reference to the presence of diluted side-stream smoke and exhaled mainstream smoke in the ambient air, and, more particularly, to the public controversy over the effect such smoke has, if any, on non-smokers who are exposed to it. Industry critics contend that exposure to ETS both causes disease in non-smokers and exacerbates existing health conditions for individuals who suffer from heart and lung disease.

A great deal of public attention has recently been focused on efforts to curb smoking in the workplace, in restaurants and other public accommodations, and in airplanes and other forms of public transportation. There also has been a good deal of press attention given to the claim that parents harm their children by smoking at home. It is likely that questions might arise during an appearance by company executives before the congressional committees currently considering smoking and health issues about the industry's position on the health effects of ETS.

The question of whether ETS exposure is a health risk for non-smokers is often not decided on a scientific basis; rather politics, speculation, and emotion-laden arguments

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have replaced open scientific discussion of ETS' alleged health effects. In a number of instances, investigators have simply assumed that ETS is a health risk and have attempted to estimate the magnitude of the assumed risk. Such reports claim that hundreds or even thousands of non-smokers die of diseases due to ETS. However, these investigators fail to acknowledge the serious methodological problems underlying their estimates.

At several recent meetings, experts on ETS have reported the lack of valid scientific data to support such claims and the difficulty of interpreting the available scientific data on the subject. For example, the summary of a report on an international workshop held at the University of Geneva in March 1983, contains the statement, "an overall evaluation based upon available scientific data, leads to the conclusion that an increased lung cancer risk for non-smokers from ETS exposure has not been established."[1]

Likewise, the organizer of an international symposium on the medical perspectives of ETS, organized and sponsored by United States and European private and governmental bodies, and held in Vienna, Austria, in 1984 notes:

> Summarizing all the results presented here, I come to the conclusion that up to now there is no scientific evidence of a causal relationship between passive smoking and lung cancer. In my opinion, all the epidemiological studies carried out so far lack the appropriate methods to determine the extent of exposure to passive smoking. So, I do not think any epidemiological investigation will be performed

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> convincingly in the future until the problem of determining the extent of exposure to passive smoking has been solved satisfactorily.[2]

A 1983 U.S. Government sponsored review of research on possible respiratory effects of exposure to ETS concluded:

> A review of the data from the studies which have been carried out or are in progress which address the effect of passive smoking on the respiratory system suggests that the effect varies from negligible to quite small. From this review, it was not possible to determine whether there is a specific group which is at increased risk or what the mechanisms of the effect (if any) may be.[3]

In 1986, the Surgeon General published a report on ETS which concluded that ETS is harmful to non-smokers. It specifically reported that ETS exposure causes lung cancer. Close examination of this report, however, shows that the conclusion is based on studies which even the report acknowledges to have serious methodological flaws. The report also acknowledges the need for more accurate data on exposure than simply using spouse's smoking habits.

On the question of the possible effects of ETS exposure on the adult respiratory function, the report concedes:

> The physiologic and clinical significance of the small changes in pulmonary function found in some studies of adults remains to be determined. The small magnitude of affect implies that a previously healthy individual would not develop chronic lung disease solely on the basis of involuntary tobacco smoke exposure in adult life.

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With regard to ETS and acute respiratory illness, the report notes "there are no studies of acute respiratory illness experienced in adults exposed to environmental cigarette smoke." The report also acknowledges that lack of data to support claims that exposure to ETS can induce asthmatic attacks in sensitive individuals. It notes that studies to date used artificial test settings, unrealistically high levels of smoke, and inadequate control of psychological factors.

The report concludes the exposure to ETS has not been shown to contribute to cardiovascular disease or to be hazardous to patients with pre-existing heart disease:

> Further studies on the relationship between involuntary smoking and cardiovascular disease are needed in order to determine whether involuntary smoking increases the risk of cardiovascular disease.

The studies cited by industry critics as evidence of health claims from ETS exposure contain serious methodological inadequacies. Questions have arisen because in no epidemiological study to date has the alleged risk to non-smokers been assessed by actual measurements of ETS in the home, in the workplace, or in public. Other problems arise because of the failure to account and control for the many factors associated with the diseases in question. Such factors commonly overlooked include diet, lifestyle, heredity, and the wide variety of chemical, physical, and biological contaminants common to the modern indoor and outdoor environment. Failure

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to account and control for these factors makes interpretation of study results questionable.

An American researcher, for example, examined the epidemiological studies on ETS and lung cancer for possible bias and error which could explain the reported increased risk among non-smoking women married to smokers. He concluded:

> In summary, when a spouse's smoking status is used to estimate a non-smoker's ETS exposure, 'a considerable amount of misclassification' may result. Since selection bias and confounding must also be considered, extreme caution is required in interpretation of these studies. This is especially so when [the] literature as a whole contains several studies reporting no significant association between ETS exposure and lung cancer, as well as various inconsistencies, both among and within studies.[4]

In a paper presented to the U.S. Air Pollution Control Association meeting in 1986, American researchers examined the epidemiological data on ETS and lung cancer and concluded:

> The epidemiological data concerning any cause and effect relationship between ETS and lung cancer is, at best, equivocal. The most persistent weakness is the absence of adequate exposure information. This precludes the evaluation of those response relationships -- a critical element of the test for causality. Moreover, the problems of sampling bias and misclassification complicate interpretation of studies done to date.[5]

Critics argue that asthmatics and individuals with pre-existing heart and lung diseases are particularly vul-

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nerable to various environmental influences, including ETS. Reviews on the health effects of environmental tobacco smoke exposure, however, report that the data is limited and conflicting on this subject.

The Surgeon General's 1984 report on chronic obstructive lung disease contains a chapter on ETS which concludes, among other points, that "the limited existing data yield conflicting results concerning the relationship between passive smoke exposure and pulmonary function changes in patients with asthma." The report further explained:

> There are very limited data on the effects of passive smoke exposure in patients with pre-existing pulmonary disease, and the available data are conflicting. Clinical studies have suggested a relationship between respiratory symptoms in asthmatics and exposure to parental cigarette smoke, but methodologic problems complicate the interpretation of the limited available data.

The 1986 Surgeon General's Report on ETS similarly acknowledges conflicting data on ETS exposure in children and adults with asthma. On children it states:

> Epidemiologic studies of children have shown no consistent relationship between the report of a doctor's diagnosis of asthma and exposure to involuntary smoking. Although one study showed an association . . . , others have not.

On the question of whether or not ETS exposure affects adults with asthma, the report reviewed three studies on the subject

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and acknowledged conflicting results and methodological problems. It states:

> Acute exposure in a chamber may not adequately represent exposure in the general environment. Biases in observation and (in the). . . selection of subjects and the subject's own expectations may account for the widely divergent results. Studies of large numbers of individuals with measurement of the relevant physiologic and exposure parameters will be necessary to adequately address the effects of environmental tobacco smoke exposure on asthmatics.

The plausibility of the claim that ETS exacerbates symptoms in those with existing cardiovascular disease rests on the observations that ETS contains carbon monoxide and nicotine. Actual measurements, however, suggest that carbon monoxide and nicotine in the ambient air do not substantially increase levels of these substances in the blood of non-smokers.[6]

The evidence linking parental smoking and children's health problems is contradictory and weak. There is no scientific agreement that the reported associations are in fact due to parental smoking and not to a variety of so-called confounding factors such as socio-economic status, and the use of gas cookers and heaters. Surveys and studies on this subject have produced conflicting results.[7] Some studies report that children whose parents smoke have more respiratory symptoms and illnesses than those whose parents are non-smokers. Others report no such effects.

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Although some individuals are annoyed by the sight and smell of tobacco smoke and a few even report experiencing irritation, the existence of human allergens in tobacco smoke has not been established.[8] In many cases, the sensitive individual may be responding to high room temperatures, lack of ventilation, or even to the mere sight of tobacco smoke. A review of the scientific literature on the subject indicates that it must be questioned what people really mean when they say they are "allergic" to tobacco smoke. They may simply not like the sight and smell of tobacco smoke and interpret this to mean that they are "allergic" to tobacco smoke.

Tobacco smoke is only one of many possible contributors to indoor air quality. Other suspected factors affecting indoor air quality include allergens such as dust mites and spores from fungi, and chemicals such as formaldehyde, ozone, and nitrogen dioxide.[9] To ban one suspected constituent because it is visible and commonplace is unjustifiable. Reaction to tobacco smoke may merely indicate a more general ventilation problem.

The alleged health hazards of ETS exposure in the workplace have also received considerable attention in the media. There is very little data on ETS exposures at the workplace; nonetheless, some reports strongly recommend workplace smoking restrictions. These recommendations are often

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implicitly based on the desire to force smokers to reduce their smoking, rather than on any sound scientific data.

Scientists who have studied the air quality in office buildings have concluded that tobacco smoke does not play a major role in the quality of indoor air.[10] It has been reported that neither pollution levels nor complaints about "stale" air are higher in buildings where smoking is permitted than in buildings where it is prohibited.[11] Although smoking restrictions applying to offices and public places are being considered, none to date is based on actual air quality studies. When actual measurements in real life situations are made, the results suggest that ETS is not a significant factor of indoor air quality, and that smoking regulations are unnecessary to assure adequate indoor air quality.[12] Current studies suggest that inadequate ventilation is the most important factor determining indoor air quality.[13]

Since the data on ETS are inconclusive, public smoking laws constitute unnecessary government intervention in matters of individual behavior. To the extent that scientific debate on ETS is suppressed and data misused, the public may accept coercive or punitive laws it would not otherwise accept. There is a need for balanced discussion on ETS. Some of the press coverage given to ETS issues tends to highlight the most alarming or sensational claims, often without regard to the critical limitations of the methodologies and

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data used in the studies.

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NOTES

1. Rylander, R. et al. (Ed.), ETS--Environmental Tobacco Smoke, Report from a Workshop on Effects and Exposure Levels, March 15-17, 1983, University of Geneva, Switzerland, published in European Journal of Respiratory Diseases (1984) Supplementum No. 133, Vol. 65, page 144.

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3. U.S. Department of Health and Human Services, Report of Work Shop on Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies, May 1-3, 1983, Public Health Service, National Institute of Health (December 1983).

4. Lee, P.N. et al., "Relationship of Passive Smoking to Risk of Lung Cancer and Other Smoking Associated Diseases," British Journal of Cancer (1986) 54:167.

5. Balter, N.J. et al., "Causal Relationship Between Environmental Tobacco Smoke and Lung Cancer in Non-Smokers: A Critical Review of the Literature," paper presented at the 79th Annual Meeting of the U.S. Air Pollution Control Association, Minneapolis, Minnesota, June 22-27, 1986, p. 13.

6. Schievelbein, H. & Richter, F., "The Influence of Passive Smoking on the Cardiovascular System," Preventive Medicine (November 1984) 13:626.

7. U.S. Surgeon General, The Health Consequences of Smoking--Chronic Obstructive Lung Disease (1984), pp. 387-389, 392, 401; U.S. Department of Health and Human Services, Report of Workshop on Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies, May 1-3, 1983, Public Health Service, National Institute of Health (December 1983) p. 3; U.S. National Research Council, Environmental Tobacco Smoke--Measuring Exposures and Assessing Health Effects, prepared by the NRC Committee on Passive Smoking, Board on Environmental Studies and Toxicology (1986), National Academy Press, Washington, D.C.

8. See Berglund, B., et al. (Eds.), Proceedings of the Third International Conference on Indoor Air Quality and Climate, held in Stockholm, Sweden, August 20-24, 1984.

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9. Id., Andersen, I. & Korsgaard, J. "Asthma in the Indoor Environment--Assessment of the Health Implications of High Indoor Air Humidity," pp. 79-86.

10. Sterling, T.D. et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments," Journal of Occupational Medicine (January 1987), 29:57.

11. "Statement of Gray Robertson, President, ACVA Atlantic, Inc.," (December 10, 1987), Statement Accompanying Tobacco Institute press release.

12. Sterling, T.D. et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments," Journal of Occupational Medicine (January 1987), 29:61.

13. Id.

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PRIOR TOBACCO INDUSTRY POSITIONS ON SMOKING AND HEALTH

This paper reviews the positions taken by the tobacco industry on smoking and health issues, both in testimony before Congress and in publications and programs of The Tobacco Institute. Not surprisingly, the positions of the industry have changed to some extent over the years as the scientific studies and debate on smoking and health have evolved. This paper merely outlines these positions and notes some of the specific statements that may be challenged by our opponents in future legislative hearings.

In general, the industry has taken a consistent position that proof is lacking of adverse health effects from smoking, passive smoking, or any of the constituents found in tobacco smoke. Industry research expenditures have generally been characterized as substantial, more generous than those of other private groups, and having no restrictions. In addition the industry has denied any addictive qualities of smoking. Finally, advertising of cigarettes has been stated to be merely related to brand preferences, and to have no effect on recruiting youth, or non-smokers, to smoke.

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I. WHETHER SMOKING CAUSES DISEASE

The industry has consistently taken the position that it is an "open question" whether there is a causal link between smoking and various diseases, and that further research is necessary. This position has been maintained throughout the various congressional hearings and industry publications.

Industry members have admitted that statistical associations exist between smoking and certain diseases, but they have emphasized that such a statistical relation does not establish causation. Industry spokespeople have consistently denied that there is sufficient proof of a causal link between smoking and lung cancer; cardiovascular disease (including stroke and high blood pressure); chronic obstructive pulmonary diseases (including emphysema and bronchitis); cancers of the pancreas, larynx, esophagus, bladder, and urinary tract; oral and pharyngeal cancers; ulcers (including peptic ulcers); and overall mortality rates. The industry has similarly criticized the claim that there are 300,000 excess deaths per year due to smoking, characterizing this claim as "sheer speculation." See, e.g., The Cigarette Controversy, Eight Questions and Answers 26.

There have been similar statements on the effect of smoking on women, especially during pregnancy. The

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industry has acknowledged that "smoking mothers, on the average, have slightly lighter weight babies." The Cigarette Controversy 8 (1974). But the industry has denied that there is proof of a causal link to the following conditions which have been statistically linked to tobacco use: increased perinatal mortality, abortion, stillbirth, birth defects, early menopause, decreased child development, disease in those using oral contraceptives.

The industry has likewise stated that there is no scientific evidence linking passive smoking to any disease. Statements on passive smoking tend to be stronger than those regarding primary smoking. See, e.g., Testimony of Horace Kornegay 633 (1972) (claim "seems extremely far-fetched"). The questioning of adverse health effects includes questioning any effect on children of smoking parents. Cf. Women and Smoking 7 ("Is it true that smoking mothers can harm their children by smoking around them? Studies in this area report conflicting results.").

There have been a number of strong statements denying causal links. Dr. Clarence Little, in 1969 written testimony as Scientific Director of the Council for Tobacco Research ("CTR") (April/May hearings), said that "[i]f anything, the pure biological evidence is pointing away from, not toward, the causal hypothesis."

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In 1978 Horace Kornegay testified that "there has been no highly qualified, reputable person who has made a statement that [smoking] affects or adversely affects the heath." A TI publication argues that "statistics can be twisted and misrepresented for scare propaganda." Fact or Fancy? 49.

There are also, however, some industry statements which may raise questions as to adverse health effects, including the following:

(1) Dr. Little, testifying for the Tobacco Industry Research Committee ("TIRC") at 1957 congressional hearings, stated that any chronic irritation "is a bad risk in any form of cancer" and that "inhalation of smoke" is an irritant. Dr. Little also referred during his testimony to an article in which he said "it would seem unwise to fill the lungs repeatedly with the suspension of fine particles of tobacco products of which smoke consists."

(2) Industry statements have acknowledged that smoking has a role "in depressing ciliary mobility" but have denied that this has any proven relation to the development of lung cancer. See, e.g., Testimony of Dr. Robert Hockett 810 (1965 - March/April hearings.).

(3) Joseph Cullman, speaking as Chairman of the Executive Committee of TI at 1969 congressional hearings (April/May hearings), stated that "Caution:

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Introduction of polluted air into lungs, including cigarette smoke, is injurious to health" was "a fair statement," but then also said "I don't think people really know" if smoking is injurious.

(4) Dr. Robert Hockett, in October 5, 1978 testimony, stated that it is a "plausible possibility" that smoking has "an effect on small airways"; that smoking causes "a certain amount of irritation"; that for a small group of persons who "for genetic reasons are highly susceptible" to chronic pulmonary disease" smoking can be "a grave danger"; and that smoking reduces muscle and tissue production in youth.

(5) In May 1983 congressional testimony Dr. Sheldon Sommers stated that those with preexisting emphysema should not smoke and that for them smoking "is going to cause difficulty."

In addition the industry has stated that "[y]oung people should not smoke." Fact or Fancy? 51. In October 5, 1978 congressional testimony, Dr. Robert Hockett similarly stated that nicotine "and perhaps other ingredients in smoke reduced somewhat the efficiency of the production of muscle and other body tissues" in youth, such that smoking could cause harm absent adjustments to diet.

There has also been some conflict as to whether smoking can cause an allergic reaction. Thus, Science

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and Smoke, (1978), quotes testimony that "it has not been clearly established that allergens for man are present in tobacco smoke." A TI newsletter, however, merely states that "True tobacco allergy . . . is rare." Smoking and the Public 5. A CTR report, introduced at 1965 congressional testimony (April/May hearings), notes research that "certain tobacco effects . . . may be due to specific allergic susceptibility of particular individuals," and an exhibit to testimony in 1972 by Horace Kornegay states that "there may be people who have an unusual hypersensitivity to tobacco smoke."

As noted above, the industry has acknowledged that there is a statistical link between smoking and various diseases, even if statistics do not prove causation. In some instances, however, even the statistical link of smoking to disease has been disputed. For example in The Cigarette Controversy: Why More Research is Needed 9 (Feb. 1984) statistical links to "perinatal problems, including low birth weight, fetal growth retardation, perinatal mortality and congenital abnormality" are questioned in light of recent studies. See also Testimony of Dr. Sheldon Sommers 1080 (1969 - April/May hearings) ("evidence both favoring and opposing" statistical links).

Some industry statements also argue that if a causal relation of cigarettes to disease were ever

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established, the industry would take action to modify its product: "If one or more of these compounds, as found in smoke, can be proved harmful, modern technology certainly would be applied in efforts to modify the product accordingly." The Cigarette Consumer Controversy 13 (Jan. 1981). See also Testimony of Bowman Gray 160 (1964) (if harmful effects were found "We get awfully fast to work to see what we can do about it."); Testimony of George Allen 943 (1965 - March/April hearings) ("If there is something in tobacco that is causally related to cancer or any other disease, the tobacco industry wants to find out what it is"). As Dr. Little stated at the 1957 congressional hearings, "I don't think there is one of them [i.e., tobacco executives] that is stupid enough to want to bluff for a minute" regarding any adverse health effects.

In responding to these criticisms, the industry has noted that there are a variety of other factors which may be causal agents for the diseases that have been statistically linked to smoking. Most emphasis has been on the role of heredity and personality, as well as the role of viruses and of environmental pollution. See, e.g., The Answers We Seek and The Need to Know, (two TI films). One often-cited position is that it is not smoking that causes disease, but rather the underlying personality of those who are most likely to

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smoke. See, e.g., Fact or Fancy? 29 ("It may be the smoker rather than the smoking that should be investigated").

Finally, the industry sometimes noted the potential benefits of smoking, particularly in the area of stress reduction. One publication in particular, The Smoking Controversy: A Perspective (Dec. 1978), argues strongly for the beneficial effects of smoking, including the risk that some persons might be subject to "critical levels of hypertension" if they could not smoke. Dr. Robert Hockett, in October 5, 1978 congressional testimony, similarly notes benefits from smoking in the areas of relieving anxiety and tension, aiding in digestion, and reducing the number of cases of Parkinson's disease and aphthous ulcers. Testimony of Dr. Sheldon Sommers on March 12, 1982 notes beneficial effects of smoking for those with "severe psychic or psychologic problems."

II. INDUSTRY RESEARCH EFFORTS

The industry's descriptions of its research efforts usually consist of statements regarding the amount of money spent on research (either during the prior year or since the establishment of CTR) and the names of recipients of grant money. Industry spokespersons also consistently stress that the grantees

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have complete freedom in their research and in the decision whether to publish the results. The research funding is usually described as "no-strings attached," with "complete freedom and autonomy" given to the researchers.

The nature of the research is also usually described only in general terms. Some publications have described tobacco industry research as directed at "smoking and health," and congressional testimony has used similar phrases, such as "the relationship between tobacco use and health." Among the stronger statements is one (repeated on several occasions by Dr. Sheldon Sommers) that from 1970 to 1982 $14 million was spent on "whether cigarette smoking causes lung cancer in animals," and the results "proved negative." Dr. Sommers also stated in March 1983 testimony that two-thirds of CTR's research grants were spent on cancer research. In 1972 testimony Dr. Sommers, speaking as Chairman of CTR, noted that grants are awarded based on "competitive scientific merit and relevance of the proposals received to smoking and health problems." Nevertheless Dr. Little, in 1957 Congressional testimony, stated that grant money is given out in a "very, very broad" range of areas "not confined to the effects of tobacco or any tobacco product." Dr. Robert Hockett of CTR stated in 1969 testimony (April/May

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hearings) that CTR is "concentrating study on the diseases that have been reported to be associated statistically with tobacco use."

Industry statements have also criticized research by private organizations active in anti-smoking work, such as the American Cancer Society. One paper characterizes the research of these organizations by saying that "there is good reason to suspect waste and misdirection." The Smoking Controversy: A Perspective 19 (Dec. 1978). The usual citations are more mild, however, and merely note that the tobacco industry has spent more on smoking and health research than any of the other organizations interested in these issues, such as the American Cancer Society.

III. INGREDIENTS/CONSTITUENTS OF SMOKE

The industry has stated that no constituent of tobacco smoke has been found to be a cause of cancer in humans in the amount in which it is present in smoke. The denial of any harmful effect has encompassed the following substances: "tar," nicotine, carbon monoxide, benzpyrene, nitosamines and nitrogen oxides, hydrogen cyanide, and beta naphthylamine.

The precise formulation of this positions varies. Most often, the industry has made the general statement that the concentrations of these substances in smoke are

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so minute as to be inconsequential, and that only sophisticated research tools can detect these constituents. Cf. Testimony of Dr. Robert Hockett 105 (1972) (cigarette smoke is "about 2 or 3 percent carbon monoxide by volume"). At other times, the focus is on the absence of any smoke inhalation studies finding adverse health effects, or the absence of studies on primates. For nicotine there have been further statements that not only is nicotine not harmful, but also that it may have beneficial effects, including that it "stimulates the learning process." Nicotine's effect is often paralleled to that of "mild exercise." See, e.g., Testimony of Dr. Robert Hockett 1112 (1969 - April/May hearings) ("effects similar to those of light exercise on the work of the heart").

Statements regarding "tar" focus on arguments that "tar" is not actually an ingredient in smoke, and that analysis of it is not an accurate method of research. Similarly there is repeated criticism of skin-painting of "tar" as a method of research, since such studies "involve the wrong material (that is the condensate instead of whole smoke), in the wrong dose, and in the wrong form, applied to the wrong tissue of the wrong animal." Testimony of Dr. Robert Hockett 102 (1971).

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By contrast Dr. Little, at the 1957 congressional hearings, admitted benzpyrene is carcinogenic, although only "[i]f you get enough of it at the right place." In addition there is at least one reference in congressional testimony to the presence of insecticides and pesticides on cigarettes. Joseph Cullman, speaking at 1969 Congressional hearings (April/May hearings), stated that it was unclear "what if anything of these insecticides or pesticides get through to the products."

Statements regarding the ingredients used in the manufacture of cigarettes, as opposed to the natural constituents of tobacco smoke, are rare. Beyond broad references to supplying information to the government, specific ingredients are not mentioned, and their health effects are not discussed. Instead, there are broad statements that no causal link between disease and any cigarette ingredient has been proven. The distinction between ingredients and constituents is often blurred in these statements. See, e.g., Testimony of Bowman Gray 251 (1965 - March/April hearings) ("have not been able to establish that any particular ingredient or compound in cigarette smoke is the cause of illness.").

IV. ADDICTION/WHY PEOPLE SMOKE

There are very few industry statements on the issue of addiction. To the extent such statements

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exist, they deny any addictive effect. See, e.g., Testimony of Edward Horrigan 390 (1982 - March 5, 11, and 12 hearings) ("absolutely no proof that cigarettes are addictive"); Testimony of Dr. Robert Hockett 22 (1978 - October 5 and 6 hearings) ("there is an adjustment" to smoking over time and issue of dependence is "a very tough question").

More often there are statements regarding why people smoke, and in particular why young people begin to smoke. In this regard the industry position has been that young people begin to smoke primarily because of peer pressure, and other social influences, and not as a result of advertising. See, e.g., Testimony of Horace Kornegay 190 (1978 - February hearing) ("I do not believe . . . that cigarette advertising induces young people to smoke"); On Tobacco: 21 Questions and Answers 7 ("complex psychological, cultural and socioeconomic factors.") The industry explicitly states that it is not aiming its products at youth.

V. ADVERTISING AND PROMOTIONAL ACTIVITY

Industry statements in this area focus on the argument that the claimed harmful effects from smoking have been widely stated and are known to more than 90% of the population. See, e.g., Testimony of William Kloepfer 674 (1969 - April/May hearings) ("Never has any

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product been so extensively publicized with an increasing crescendo of inflammatory insistence that its use is hazardous.") Conversely, Mr. Cullman has stated that reductions in advertising do not decrease consumption of cigarettes. See, e.g., Testimony of Joseph Cullman 114 (1969 - July hearings) ("[t]here isn't any evidence around the world that reduction in advertising or discontinuance of advertising will materially affect cigarette consumption"). Finally, the industry position has been that smoking is an adult habit, and that advertisements should not, and do not, be directed towards young people.

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Statement of Charles O. Whitley

on behalf of

The Tobacco Institute

to the

Subcommittee on Transportation, Tourism, and
Hazardous Materials of the
Committee on Energy and Commerce
United States House of Representatives

May 4, 1988

Mr. Chairman, distinguished members of the Subcommittee and former colleagues. The letter inviting us to participate in this hearing indicated that the Subcommittee was interested in receiving a report concerning operation of The Tobacco Institute Testing Laboratory ("TITL"). The letter indicated, more specifically, that the Subcommittee wanted further information concerning (1) the relationship of The Tobacco Institute to TITL, (2) how TITL tests cigarettes for "tar" and nicotine and (3) the relationship between the testing methods used by TITL and the testing methods that were employed by the Federal Trade Commission's cigarette testing laboratory. I am accompanied by Dr. William L. Steele, Director of The Tobacco Institute Testing Laboratory. Dr. Steele and I will attempt to provide the information the Subcommittee is seeking in these areas.

A number of issues that were thought to be raised by the February 1987 closing of the Federal Trade Commission's

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cigarette testing laboratory were addressed at a hearing before this Subcommittee on May 7, 1987. The Commission advised the Subcommittee at that time that the Commission decision was based on budgetary considerations. We assured the Subcommittee that the Commission decision was not prompted by the tobacco industry, and that closing of the Commission laboratory would not have any negative or undesirable effects. For reasons that I will explain in a moment, our experience over the past year should remove any doubt -- from any quarter -- of the Commission's continued ability to ensure the accuracy of any advertised "tar" and nicotine ratings.

With your permission, Mr. Chairman, I would like to address in turn each of the issues or questions that were posed in the letter inviting us to appear today. The Tobacco Institute Testing Laboratory, or TITL, was created in 1966 by the cigarette manufacturers that were members of The Tobacco Institute. For administrative or bookkeeping purposes only, TITL was organized as part of The Tobacco Institute. As a practical matter, however, TITL operates as an independent entity. The Director of TITL, Dr. Steele, reports to a committee of scientific representatives of the cigarette manufacturers. That committee, in consultation with Dr. Steele, develops and approves TITL's annual budget and is ultimately responsible for resolving any issues that arise in connection with TITL's operations.

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Contrary to the impression apparently held by many, the basic methodology that has been used for the past twenty years to test cigarettes for "tar" and nicotine was not developed by either the Federal Trade Commission or TITL. In April 1964, a major study on cigarette testing was published by Dr. Clyde L. Ogg, a scientist employed by the Department of Agriculture. The study described a methodology for testing for "tar" and nicotine -- the so-called "Cambridge Filter Method."*/ Following discussions with the cigarette manufacturers and public hearings, the Commission adopted the Cambridge Filter Method, without significant change, for the testing laboratory that it had decided to establish. See 32 Fed. Reg. 11,778 (1967). The Cambridge Filter Method was utilized by the Commission laboratory until the closing of that laboratory in February 1987.

From the time of its creation in November 1966 through the most recent test cycle, TITL has utilized the same testing methodology as the Commission laboratory. During the approximately 20-year period when both laboratories were operating, Commission and TITL test results differed significantly on only two occasions. In 1976, TITL staff discovered that operator oversight had led the Commission laboratory to overstate "tar" and nicotine for several cigarette brands. In

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*/ Ogg, "Determination of Particulate Matter and Alkaloids (as Nicotine) in Cigarette Smoke," 47 J. Ass'n Official Agricultural Chemists 356 (1964).

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1980, when the Commission and TITL first began to test for carbon monoxide, TITL staff found that the Commission laboratory had overstated its CO results due to a programming error. In both instances, the Commission laboratory ultimately revised its figures so that they matched TITL's test results.

When the Commission decided in February 1987 that it could no longer justify the cost of maintaining its laboratory in view of the testing being performed by the cigarette companies and TITL, Commission representatives initiated discussions with the cigarette manufacturers looking toward an arrangement whereby a Commission representative would be permitted to monitor testing by TITL. Under the arrangement requested by the Commission, and agreed to by the cigarette manufacturers, the Commission must be notified at least ten days before the commencement of any cigarette market pickup, which is the first step in any test cycle. It is understood that the Commission representative will review at that time all aspects of the procedures to be used in the pickup. He also may review any documents that discuss the pickup procedures, including any contracts that TITL has entered into with the firm selected to perform the pickup.

Once the cigarettes to be tested begin to arrive at TITL, the Commission representative is guaranteed an essentially unlimited right of access to the TITL test facility. Under the terms of the agreement between the Commission and the cigarette manufacturers, the Commission representative may

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visit TITL "on any schedule he deems appropriate" without prior notice to Dr. Steele or any other TITL staff member. In addition, the agreement guarantees the Commission representative complete access to any TITL computer sheets and other test documents that are generated in the course of any test cycle. I would like to submit for the record a copy of the correspondence describing the terms of the monitoring agreement.

At the hearing held by this Subcommittee on May 7, 1987, the Commission representatives stated that they were satisfied that the Commission would be able to continue to ensure the accuracy of the "tar" and nicotine figures included in cigarette advertising. Since that earlier hearing, TITL has completed one cigarette test cycle and has commenced a second. For both test cycles, Mr. Harold Pillsbury, who formerly was in charge of the Commission's cigarette testing laboratory, has served as the Commission's on-site representative at TITL. I can assure the Subcommittee that Mr. Pillsbury's monitoring activities have been both careful and intensive.

In fact, since the monitoring agreement has been in effect, Mr. Pillsbury has been spending from one to several days per week at TITL. He has been given complete access to TITL's files, has been permitted to participate in all aspects of the actual testing -- without limitation of any kind -- and

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has had access to test results on the same basis as TITL staff. At our suggestion, a computer hookup has even been established that permits him to work with and check the test data as they are being generated and to provide that data on a continuing basis to his colleagues at the Commission. I am not aware of a single request that Mr. Pillsbury, or any other member of the Commission staff, has made to facilitate Mr. Pillsbury's monitoring activities that has not been granted immediately and completely.

Mr. Chairman, I would like to use this opportunity to address two additional points relating to the Commission laboratory that have been raised at various times in the past. When the Commission decided to close its cigarette testing laboratory, some observers suggested that the cigarette manufacturers supported, or had sought, the laboratory's closing. A suggestion also was made at that time that closing of the Commission laboratory would diminish the Commission's ability to refine or improve the methodology used to test for "tar" and nicotine. As we have attempted to explain on other occasions, both suggestions are without merit.

The fact is that the tobacco industry opposed the Commission's plan to close its cigarette testing laboratory. That opposition was expressed at meetings with Commission staff as early as October 29, 1984, when the possible closing of the laboratory was first raised, and continued without

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change until the decision was announced. In fact, an effort was made on behalf of the tobacco industry even after the closing decision was announced to convince the Commission to reverse its decision.

I should emphasize that the tobacco industry's opposition to closing of the Commission laboratory stemmed largely from concern that any closing of the Commission laboratory would be misconstrued. We hope that this hearing will serve to eliminate any lingering misapprehensions in this area.

Finally, I would like to comment briefly on the suggestion that closing of the Commission's cigarette testing laboratory has reduced, or may reduce in the future, the Commission's ability to refine or improve the methodology used to test for "tar" and nicotine. I addressed earlier one of the major misconceptions that underlies that suggestion. As noted, the Commission laboratory did not play a significant role in development of the methodology currently being used to test for "tar" and nicotine. In addition, from the beginning, whenever questions have arisen concerning possible refinements in that methodology, the Commission has relied upon outside experts and contract laboratories -- in particular, the Department of Energy's testing facility at Oak Ridge, Tennessee. Those facilities are as available to the Commission today as they have been in prior years.

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In summary, Mr. Chairman, both we and the Commission have made every effort to satisfy the concerns that were expressed at the hearing on May 7. We have developed a good and productive working partnership with the Commission in testing cigarettes for "tar," nicotine and CO. As a result of that partnership, the Federal Government has realized substantial cost savings without reducing, in the slightest, the Commission's ability to satisfy its responsibilities.

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THE HEALTH CONSEQUENCES OF INVOLUNTARY SMOKING

a report of the Surgeon General

1986

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Public Health Service
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Center for Health Promotion and Education
Office on Smoking and Health
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DEPARTMENT OF HEALTH AND HUMAN SERVICES
WASHINGTON, D.C. 20201

DEC 5

The Honorable Thomas P. O'Neill, Jr.
Speaker of the House
of Representatives
Washington, D.C. 20515

Dear Mr. Speaker:

It is my pleasure to transmit to the Congress the 1986 Surgeon General's Report on the health consequences of smoking, as mandated by Section 8(a) of the Public Health Cigarette Smoking Act of 1969. The current volume, entitled The Health Consequences of Involuntary Smoking, examines the scientific evidence on the health effects resulting from nonsmoker exposure to environmental tobacco smoke.

The issue of whether or not tobacco smoke is carcinogenic for humans was conclusively resolved more than 20 years ago when the first report on smoking and health was issued in 1964. Based on the current report, the judgment can now be made that exposure to environmental tobacco smoke can cause disease, including lung cancer, in nonsmokers. It is also clear that simple separation of smokers and nonsmokers within the same airspace may reduce but cannot eliminate nonsmoker exposure to environmental tobacco smoke.

The report also reviews an extensive body of evidence which establishes an increased risk of respiratory illness and reduced lung function in infants and very young children of parents who smoke. This effect is more pronounced if both parents smoke than if only one parent smokes. As a physician, I believe that parents should refrain from smoking around small children both as a means of protecting their children's health and to set a good example for the child.

Today, only 30 percent of the adult population in the United States are smokers--the lowest level of smoking in the country since World War II, reflecting that the great majority of the population has never smoked or has successfully quit.

Accompanying this decline in overall prevalence of cigarette smoking has been an increased concern for protecting the health and well being of nonsmokers, as evidenced by the number of laws and regulations restricting smoking in public places. Today, 40 States and the District of Columbia have enacted some form of legislation to restrict smoking in public. Increasingly, these laws pertain to protecting nonsmokers in many different settings, including the workplace.

Based on the evidence presented in this report, the choice to smoke should not interfere with the nonsmoker's choice for an environment free of tobacco smoke.

Sincerely,

Otis R. Bowen M.D.

Otis R. Bowen, M.D.
Secretary

Enclosure

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CHAPTER 1

INTRODUCTION, OVERVIEW, AND SUMMARY AND CONCLUSIONS

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- Policies Restricting Smoking in Public Places and the Workplace

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Development and Organization of the 1986 Report

The 1986 Report was developed by the Office on Smoking and Health of the U.S. Department of Health and Human Services as part of the Department's responsibility, under Public Law 91-222, to report new and current information on smoking and health to the United States Congress.

The scientific content of this Report reflects the contributions of more than 60 scientists representing a variety of disciplines. Individual manuscripts were written by experts known for their understanding of and work in specific content areas. These manuscripts were refined through a series of meetings attended by the authors, Office on Smoking Health staff and consultants, and the Surgeon General.

Upon receipt of the final manuscripts from the authors, the Office and its consultants edited and consolidated the individual manuscripts into appropriate chapters. These draft chapters were subjected to an extensive outside peer review (see Acknowledgments for individuals and their affiliations) whereby each was reviewed by up to seven experts. Their comments were integrated and the entire volume was assembled. This revised edition of the Report was resubjected to review by 17 distinguished scientists outside the Federal Government, both in this country and abroad. Parallel to this review, the entire Report was also submitted to various institutes and agencies within the U.S. Public Health Service for review and comment.

The 1986 Report contains a Foreword by the Assistant Secretary for Health, a Preface by the Surgeon General of the U.S. Public Health Service, and the following chapters:

Chapter 1. Introduction, Overview, and Summary and Conclusions
Chapter 2. Health Effects of Environmental Tobacco Smoke Exposure
Chapter 3. Environmental Tobacco Smoke Chemistry and Exposures of Nonsmokers
Chapter 4. Deposition and Absorption of Tobacco Smoke Constituents
Chapter 5. Toxicity, Acute Irritant Effects, and Carcinogenicity of Environmental Tobacco Smoke
Chapter 6. Policies Restricting Smoking in Public Places and the Workplace

Overview

Inhalation of tobacco smoke during active cigarette smoking remains the largest single preventable cause of death and disability

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for the U.S. population. The health consequences of cigarette smoking and of the use of other tobacco products have been extensively documented in the 17 previous Reports in the health consequences of smoking series issued by the U.S. Public Health Service. Cigarette smoking is a major cause of cancer; it is most strongly associated with cancers of the lung and respiratory tract, but also causes cancers at other sites, including the pancreas and urinary bladder. It is the single greatest cause of chronic obstructive lung diseases. It causes cardiovascular diseases, including coronary heart disease, aortic aneurysm, and atherosclerotic peripheral vascular disease. Maternal cigarette smoking endangers fetal and neonatal health; it contributes to perinatal mortality, low birth weight, and complications during pregnancy. More than 300,000 premature deaths occur in the United States each year that are directly attributable to tobacco use, particularly cigarette smoking.

This Report examines in detail the scientific evidence on involuntary smoking as a potential cause of disease in nonsmokers. Nonsmokers' exposure to environmental tobacco smoke is termed involuntary smoking in this Report because the exposure generally occurs as an unavoidable consequence of being in proximity to smokers, particularly in enclosed indoor environments. The term "passive smoking" is also used throughout the scientific literature to describe this exposure.

The magnitude of the disease risks for active smokers secondary to their "high dose" exposure to tobacco smoke suggests that the "lower dose" exposure to tobacco smoke received by involuntary smokers may also have risks. Although the risks of involuntary smoking are smaller than the risks of active smoking, the number of individuals injured by involuntary smoking is large both in absolute terms and in comparison with the number injured by some other agents in the general environment that are regulated to curtail their potential to cause human illness.

This Report reviews the evidence on the characteristics of mainstream tobacco smoke and of environmental tobacco smoke, on the levels of exposure to environmental tobacco smoke that occur, and on the health effects of involuntary exposure to tobacco smoke. The composition of the tobacco smoke inhaled by active smokers and by involuntary smokers is examined for similarities and differences, and the concentrations of tobacco smoke components that can be measured in a variety of settings are explored, as is smoke deposition and absorption in the respiratory tract. The studies that describe the risks of environmental tobacco smoke exposure for humans are carefully reviewed for their findings and their validity. The evidence on the health effects of involuntary smoking is reviewed for biologic plausibility, and compared with extrapolations of the risks of active smoking to the lower dose of exposure to tobacco smoke found in nonsmokers. This review leads to three major conclusions:

1. **Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers.**
2. **The children of parents who smoke compared with the children of nonsmoking parents have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures.**
3. **The simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, the exposure of nonsmokers to environmental tobacco smoke.**

The subsequent chapters of this volume describe in detail the evidence that supports these conclusions; the evidence is briefly summarized here.

Environmental Tobacco Smoke Constituents

Important considerations in examining the risks of involuntary smoking are the composition of environmental tobacco smoke (ETS) and its toxicity and carcinogenicity relative to the tobacco smoke inhaled by active smokers. Mainstream cigarette smoke is the smoke drawn through the tobacco into the smoker's mouth. Sidestream smoke is the smoke emitted by the burning tobacco between puffs. Environmental tobacco smoke results from the combination of sidestream smoke and the fraction of exhaled mainstream smoke not retained by the smoker. In contrast with mainstream smoke, ETS is diluted into a larger volume of air, and it ages prior to inhalation.

The comparison of the chemical composition of the smoke inhaled by active smokers with that inhaled by involuntary smokers suggests that the toxic and carcinogenic effects are qualitatively similar, a similarity that is not too surprising because both mainstream smoke and environmental tobacco smoke result from the combustion of tobacco. Individual mainstream smoke constituents, with appropriate testing, have usually been found in sidestream smoke as well. However, differences between sidestream smoke and mainstream smoke have been well documented. The temperature of combustion during sidestream smoke formation is lower than during mainstream smoke formation. As a result, greater amounts of many of the organic constituents of smoke, including some carcinogens, are generated when tobacco burns and forms sidestream smoke than when mainstream smoke is produced. For example, in contrast with mainstream smoke, sidestream smoke contains greater amounts of ammonia, benzene, carbon monoxide, nicotine, and the carcinogens

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2-napthylamine, 4-aminobiphenyl, N-nitrosamine, benz[a]-anthracene, and benzo-pyrene per milligram of tobacco burned. Although only limited bioassay data comparing mainstream smoke and sidestream smoke are available, one study has suggested that sidestream smoke may be more carcinogenic.

Extent of Exposure

Although sidestream smoke and mainstream smoke differ somewhat qualitatively, the differing quantitative doses of smoke components inhaled by the active smoker and by the involuntary smoker are of greater importance in considering the risks of the two exposures. A number of different markers for tobacco smoke exposure and absorption have been identified for both active and involuntary smoking. No single marker quantifies, with precision, the exposure to each of the smoke constituents over the wide range of environmental settings in which involuntary smoking occurs. However, in environments without other significant sources of dust, respirable suspended particulate levels can be used as a marker of smoke exposure. Levels of nicotine and its metabolite cotinine in body fluids provide a sensitive and specific indication of recent whole smoke exposure under most conditions.

Widely varying levels of environmental tobacco smoke can be measured in the home and other environments using markers. The time-activity patterns of nonsmokers, which indicate the time spent in environments containing ETS, also vary widely. Thus, the extent of exposure to ETS is probably highly variable among individuals at a given point in time, and little is known about the variation in exposure of the same individual at different points in time.

Lung Cancer

The American Cancer Society estimates that there will be more than 135,000 deaths from lung cancer in the United States in 1986, and 85 percent of these lung cancer deaths are directly attributable to active cigarette smoking. Therefore, even if the number of lung cancer deaths caused by involuntary smoking were much smaller than the number of lung cancer deaths caused by active smoking, the number of lung cancer deaths attributable to involuntary exposure would still represent a problem of sufficient magnitude to warrant substantial public health concern.

Exposure to environmental tobacco smoke has been examined in numerous recent epidemiological studies as a risk factor for lung cancer in nonsmokers. These studies have compared the risks for subjects exposed to ETS at home or at work with the risks for people not reported to be exposed in these environments. Because exposure to ETS is an almost universal experience in the more developed countries, these studies involve comparison of more exposed and less exposed people rather than comparison of exposed and unexposed people. Thus, the studies are inherently conservative in assessing the consequences of exposure to ETS. Interpretation of these studies must consider the extent to which populations with different ETS exposures have been identified, the gradient in ETS exposure from the lower exposure to the higher exposure groups, and the magnitude of the increased lung cancer risk that results from the gradient in ETS exposure.

To date, questionnaires have been used to classify ETS exposure. Quantification of exposure by questionnaire, particularly lifetime exposure, is difficult and has not been validated. However, spousal and parental smoking status identify individuals with different levels of exposure to ETS. Therefore, investigation has focused on the children and nonsmoking spouses of smokers, groups for whom greater ETS exposure would be expected and for whom increased nicotine absorption has been documented relative to the children and nonsmoking spouses of nonsmokers.

Of the epidemiologic studies reviewed in this Report that have examined the question of involuntary smoking's association with lung cancer, most (11 of 13) have shown a positive association with exposure, and in 6 the association reached statistical significance. Given the difficulty in identifying groups with differing ETS exposure, the low-dose range of exposure examined, and the small numbers of subjects in some series, it is not surprising that some studies have found no association and that in others the association did not reach a conventional level of statistical significance. The question is not whether cigarette smoke can cause lung cancer; that question has been answered unequivocally by examining the evidence for active smoking. The question is, rather, can tobacco smoke at a lower dose and through a different mode of exposure cause lung cancer in nonsmokers? The answer must be sought in the coherence and trends of the epidemiologic evidence available on this low-dose exposure to a known human carcinogen. In general, those studies with larger population sizes, more carefully validated diagnosis of lung cancer, and more careful assessment of ETS exposure status have shown statistically significant associations. A number of these studies have demonstrated a dose–response relationship between the level of ETS exposure and lung cancer risk. By using data on nicotine absorption by the nonsmoker, the nonsmoker's risk of developing lung cancer observed in human epidemiologic studies can be compared with the level of risk expected from an extrapolation of the dose–response data for the active smoker. This extrapolation yields estimates of an expected lung cancer risk that approximate the observed lung cancer risk in epidemiologic studies of involuntary smoking.

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Cigarette smoke is well established as a human carcinogen. The chemical composition of ETS is qualitatively similar to mainstream smoke and sidestream smoke and also acts as a carcinogen in bioassay systems. For many nonsmokers, the quantitative exposure to ETS is large enough to expect an increased risk of lung cancer to occur, and epidemiologic studies have demonstrated an increased lung cancer risk with involuntary smoking. In examining a low-dose exposure to a known carcinogen, it is rare to have such an abundance of evidence on which to make a judgment, and given this abundance of evidence, a clear judgment can now be made: exposure to ETS is a cause of lung cancer.

The data presented in this Report establish that a substantial number of the lung cancer deaths that occur among nonsmokers can be attributed to involuntary smoking. However, better data on the extent and variability of ETS exposure are needed to estimate the number of deaths with confidence.

Respiratory Disease

Acute and chronic respiratory diseases have also been linked to involuntary exposure to tobacco smoke; the evidence is strongest in infants. During the first 2 years of life, infants of parents who smoke are more likely than infants of nonsmoking parents to be hospitalized for bronchitis and pneumonia. Children whose parents smoke also develop respiratory symptoms more frequently, and they show small, but measurable, differences on tests of lung function when compared with children of nonsmoking parents.

Respiratory infections in young children represent a direct health burden for the children and their parents; moreover, these infections, and the reductions in pulmonary function found in the school-age children of smokers, may increase susceptibility to develop lung disease as an adult.

Several studies have reported small decrements in the average level of lung function in nonsmoking adults exposed to ETS. These differences may represent a response of the lung to chronic exposure to the irritants in ETS, but it seems unlikely that ETS exposure, by itself, is responsible for a substantial number of cases of clinically significant chronic obstructive lung disease. The small magnitude of the changes associated with ETS exposure suggests that only individuals with unusual susceptibility would be at risk of developing clinically evident disease from ETS exposure alone. However, ETS exposure may be a factor that contributes to the development of clinical disease in individuals with other causes of lung injury.

Cardiovascular Disease

A few studies have examined the relationship between involuntary smoking and cardiovascular disease, but no firm conclusion on the relationship can be made owing to the limited number of deaths in the studies.

Irritation

Perhaps the most common effect of tobacco smoke exposure is tissue irritation. The eyes appear to be especially sensitive to irritation by ETS, but the nose, throat, and airway may also be affected by smoke exposure. Irritation has been demonstrated to occur at levels that are similar to those found in real-life situations. The level of irritation increases with an increasing concentration of smoke and duration of exposure. In addition, participants in surveys report irritation and annoyance due to smoke in the environment under real-life conditions.

Determinants of Exposure

Exposure to ETS has been documented to be common in the United States, but additional data on the extent and determinants of exposure are needed to identify individuals within the population who have the highest exposure and are at greatest risk. Studies with biological markers and measurements of ETS components in indoor air confirm that measurable exposure to ETS is widespread. However, within exposed populations, levels of cotinine excretion and presumably ETS exposure vary greatly.

In a room or other indoor area, the size of the space, the number of smokers, the amount of ventilation, and other factors determine the concentration of tobacco smoke in the air. The technology for the cost-effective filtration of tobacco smoke from the air is not currently available, and because of their small size, the smoke particles remain suspended in the air for long periods of time; thus, the only way to remove smoke from indoor air is to increase the exchange of indoor air with clean outdoor air. The number of air changes per hour required to maintain acceptable indoor air quality is much higher when smoking is allowed than when smoking is prohibited.

Environmental tobacco smoke originates at the lighted tip of the cigarette, and exposure to ETS is greatest in proximity to the smoker. However, the smoke rapidly disseminates throughout any airspace contiguous with the space in which the smoking is taking place. Dissemination of smoke is not uniform, and substantial gradients in ETS levels have been demonstrated in different parts of the same airspace. The time course of tobacco smoke dissemination is rapid enough to ensure the spread of smoke throughout an airspace within an 8-hour workday. In the home, the presence of even one smoker can significantly increase levels of respirable suspended particulates.

These data lead to the conclusion that the simple separation of smokers and nonsmokers within the same airspace will reduce, but

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not eliminate, exposure to ETS, particularly in those settings where exposure is prolonged, such as the working environment.

The exposure of an individual nonsmoker to ETS is also determined by that person's time-activity pattern; that is, the amount of time spent in various locations. For adults, the duration of time spent in smoke-contaminated environments at work or at home is the principal determinant of ETS exposure, along with the levels of smoke in those environments. For infants and very young children, the smoking habit of the primary caretaker, as well as that person's time-activity pattern, is likely to play a major role in determining ETS exposure.

Policies Restricting Smoking

Policies regulating cigarette smoking with the objective of reducing explosion or fire risk, or of safeguarding the quality of manufactured products, have been in force in a number of States since the late 1800s. More recently, and with steadily increasing frequency, policies regulating smoking on the basis of the health risk or the irritation of involuntary smoking have been promulgated.

State and local governments have enacted laws and regulations restricting smoking in public places. These policies have been implemented with few problems and at little cost to the respective governments. The public awareness of these policies that results from the media coverage surrounding their implementation probably facilitates their self-enforcement. Public awareness may best be fostered by encouraging the establishment of these changes at the local level.

Policies limiting smoking in the worksite have also become increasingly widespread and more restrictive. However, changes in worksite policies have evolved largely through voluntary rather than governmental action. In a steadily increasing number of worksites, smoking has been prohibited completely or limited to relatively few areas within the worksite. The creation of a smoke-free workplace has proceeded successfully when the policy has been jointly developed by employees, employee organizations, and management; instituted in phases; and accompanied by support and assistance for the smokers to quit smoking.

This trend to protect nonsmokers from ETS exposure may have an added public health benefit—helping those smokers who are attempting to quit to be more successful and not encouraging smoking by people entering the workforce.

Summary and Conclusions of the 1986 Report

The three major conclusions of this report are the following:

1. **Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers.**
2. **The children of parents who smoke compared with the children of nonsmoking parents have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures.**
3. **The simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, the exposure of nonsmokers to environmental tobacco smoke.**

Individual chapter summaries and conclusions follow.

Health Effects of Environmental Tobacco Smoke Exposure

1. Involuntary smoking can cause lung cancer in nonsmokers.
2. Although a substantial number of the lung cancers that occur in nonsmokers can be attributed to involuntary smoking, more data on the dose and distribution of ETS exposure in the population are needed in order to accurately estimate the magnitude of risk in the U.S. population.
3. The children of parents who smoke have an increased frequency of hospitalization for bronchitis and pneumonia during the first year of life when compared with the children of nonsmokers.
4. The children of parents who smoke have an increased frequency of a variety of acute respiratory illnesses and infections, including chest illnesses before 2 years of age and physician-diagnosed bronchitis, tracheitis, and laryngitis, when compared with the children of nonsmokers.
5. Chronic cough and phlegm are more frequent in children whose parents smoke compared with children of nonsmokers. The implications of chronic respiratory symptoms for respiratory health as an adult are unknown and deserve further study.
6. The children of parents who smoke have small differences in tests of pulmonary function when compared with the children of nonsmokers. Although this decrement is insufficient to cause symptoms, the possibility that it may increase susceptibility to chronic obstructive pulmonary disease with exposure to other agents in adult life, e.g., active smoking or occupational exposures, needs investigation.
7. Healthy adults exposed to environmental tobacco smoke may have small changes on pulmonary function testing, but are unlikely to experience clinically significant deficits in pulmo-

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nary function as a result of exposure to environmental tobacco smoke alone.

8. A number of studies report that chronic middle ear effusions are more common in young children whose parents smoke than in children of nonsmoking parents.
9. Validated questionnaires are needed for the assessment of recent and remote exposure to environmental tobacco smoke in the home, workplace, and other environments.
10. The associations between cancers, other than cancer of the lung, and involuntary smoking require further investigation before a determination can be made about the relationship of involuntary smoking to these cancers.
11. Further studies on the relationship between involuntary smoking and cardiovascular disease are needed in order to determine whether involuntary smoking increases the risk of cardiovascular disease.

Environmental Tobacco Smoke Chemistry and Exposures of Nonsmokers

1. Undiluted sidestream smoke is characterized by significantly higher concentrations of many of the toxic and carcinogenic compounds found in mainstream smoke, including ammonia, volatile amines, volatile nitrosamines, certain nicotine decomposition products, and aromatic amines.
2. Environmental tobacco smoke can be a substantial contributor to the level of indoor air pollution concentrations of respirable particles, benzene, acrolein, N-nitrosamine, pyrene, and carbon monoxide. ETS is the only source of nicotine and some N-nitrosamine compounds in the general environment.
3. Measured exposures to respirable suspended particulates are higher for nonsmokers who report exposure to environmental tobacco smoke. Exposures to ETS occur widely in the nonsmoking population.
4. The small particle size of environmental tobacco smoke places it in the diffusion-controlled regime of movement in air for deposition and removal mechanisms. Because these submicron particles will follow air streams, convective currents will dominate and the distribution of ETS will occur rapidly through the volume of a room. As a result, the simple separation of smokers and nonsmokers within the same airspace may reduce, but will not eliminate, exposure to ETS.
5. It has been demonstrated that ETS has resulted in elevated respirable suspended particulate levels in enclosed places.

Deposition and Absorption of Tobacco Smoke Constituents

1. Absorption of tobacco-specific smoke constituents (i.e., nicotine) from environmental tobacco smoke exposures has been documented in a number of samples of the general population of developed countries, suggesting that measurable exposure to environmental tobacco smoke is common.
2. Mean levels of nicotine and cotinine in body fluids increase with self-reported ETS exposure.
3. Because of the stability of cotinine levels measured at different times during exposure and the availability of noninvasive sampling techniques, cotinine appears to be the short-term marker of choice in epidemiological studies.
4. Both mathematical modeling techniques and experimental data suggest that 10 to 20 percent of the particulate fraction of sidestream smoke would be deposited in the airway.
5. The development of specific chemical assays for human exposure to the components of cigarette tar is an important research goal.

Toxicity, Acute Irritant Effects, and Carcinogenicity of Environmental Tobacco Smoke

1. The main effects of the irritants present in ETS occur in the conjunctiva of the eyes and the mucous membranes of the nose, throat, and lower respiratory tract. These irritant effects are a frequent cause of complaints about poor air quality due to environmental tobacco smoke.
2. Active cigarette smoking is associated with prominent changes in the number, type, and function of respiratory epithelial and inflammatory cells; the potential for environmental tobacco smoke exposure to produce similar changes should be investigated.
3. Animal models have demonstrated the carcinogencity of cigarette smoke, and the limited data that exist suggest that more carcinogenic activity per milligram of cigarette smoke concentrate may be contained in sidestream smoke than in mainstream cigarette smoke.

Policies Restricting Smoking in Public Places and the Workplace

1. Beginning in the 1970s, an increasing number of public and private sector institutions have adopted policies to protect individuals from environmental tobacco smoke exposure by restricting the circumstances in which smoking is permitted.
2. Smoking in public places has been regulated primarily by government actions, which have occurred at Federal, State,

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and local levels. All but nine States have enacted laws regulating smoking in at least one public place. Since the mid-1970s, there has been an increase in the rate of enactment and in the comprehensiveness of State legislation. Local governments have enacted smoking ordinances at an increasing rate since 1980; more than 80 cities and counties have smoking laws in effect.

3. Smoking at the workplace is regulated by a combination of government action and private initiative. Legislation in 12 States regulates smoking by government employees, and 9 States and more than 70 communities regulate smoking in the private sector workplace. Approximately 35 percent of businesses have adopted smoking policies. The increase in workplace smoking policies has been a trend of the 1980s.
4. Smoking policies may have multiple effects. In addition to reducing environmental tobacco smoke exposure, they may alter smoking behavior and public attitudes about tobacco use. Over time, this may contribute to a reduction in smoking in the United States. To the present, there has been relatively little systematic evaluation of policies restricting smoking in public places or at the workplace.
5. On the basis of case reports and a small number of systematic studies, it appears that workplace smoking policies improve air quality, are met with good compliance, and are well accepted by both smokers and nonsmokers. Policies appear to be followed by a decrease in smokers' cigarette consumption at work and an increase in enrollment in company-sponsored smoking cessation programs.
6. Laws restricting smoking in public places have been implemented with few problems and at little cost to State and local government. Their impact on smoking behavior and attitudes has not yet been evaluated.
7. Public opinion polls document strong and growing support for restricting or banning smoking in a wide range of public places. Changes in attitudes about smoking in public appear to have preceded legislation, but the interrelationship of smoking attitudes, behavior, and legislation are complex.

CHAPTER 2

HEALTH EFFECTS OF ENVIRONMENTAL TOBACCO SMOKE EXPOSURE

Source: https://www.industrydocuments.ucsf.edu/docs/rhbj0000